PERSISTENT CAVITIES

in

PULMONARY TUBERCULOSIS.

A critical enquiry into the principles underlying causation and means of treatment; with a study of four cases.

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INTRODUCTION.

Cavitation in the lung continues to present a difficult problem in the clinical treatment of tuberculosis. The greater part of the clinical treatment of pulmonary tuberculosis is directed towards endeavouring to close cavities. This is partly due to the fact that no specific remedy has been found until recently to combat the tubercle bacillus and that in the majority of cases cavitation is present by the time the disease is diagnosed. Collapse measures in such cases may be instituted with a view to arresting the disease and also closing the cavity but only too often general rest aided by the collapse treatment results in arrest of the diease but fails to close the cavity. The cavity persists as a potential source of haemoptysis and of bronchogenic spread of the disease at an early or later date, whilst being at the same time a source of danger to the patient's family and society.

Barnes and Barnes (1928) are quoted (29) (60) as having followed the subsequent histories of 1454 patients in whom tuberculous cavities had been diagnosed. Eighty per cent. of these patients died within one year, 82% within 2 years, 85% within 3 years and 95% within 15 years; average duration of

life of 270 patients with cavities was under 16 months (29). Bacmeister (1927) is quoted (29) as giving the mortality rate of patients with cavities as 60-80%. Simpson (66)(1935) observed 1,601 cases with cavitation during a period of 14 years. Mortality rate was 64% compared with 21% in non-cavity cases at the same Sanatorium. Mortality increased with the number and size of the cavities and the length of time the cavity had persisted; thus, after the first year 41% cases were dead, after 5 years 60% and after 10 year 63% were dead. These 63% were dead as a result of tuberculosis only. Only 275 of these 1,601 cases received surgical treatment and it is not revealed how many had cavity closure as a result. At the end of the 14 year period, 86 of the cases operated on were dead. According to Kayne, Pagel and O'Shaughnessy (34)(1939) "follow-up statistics have shown that the expectation of life is greatly diminished by the presence of cavities, varying indirectly with the size of the cavity." When assessing results such as these by figures, consideration has to be given to the question whether or not active treatment has been tried. Pinner (1945) (53) points out that cavity closure is not to be expected without collapse therapy, although spontaneous closure does at times happen. Without collapse treatment patients with cavities

do not, on the average, survive long: within 2 years more than half of them have died, within 5 years this has befallen the vast majority. Pinner maintains that the differences in opinion regarding prognosis with cavitation and the need for collapse therapy depend largely on personal experience. Some clinicans see many old cases where spontaneous cavity closure has occurred, whilst the majority see the earlier cases with higher mortality rates in untreated or unsuccessfully treated cavities.

Originally a cavity was regarded as little more than a hole in the lung as a result of excavation of necrosed tissue. Within recent years it has come to be recognised that inflation from the bronchus plays an important role. In 1922, Hall (30) reported on a case where a cavity, judged by physical signs, simulated a pneumothorax. At post-mortem the appearances suggested that a cavity had been inflated and distended grossly by a valve mechanism within a damaged draining bronchus. No such valve was demonstrated however. In 1930, Pearson (50) reported a case in which he was able to demonstrate by introducing a needle into the cavity through the chest wall, that there was an intracavitary pressure + 4 + 16. Upon withdrawing 750 cc. air pressures were zero + 9. Pearson was probably one of the earliest observers in this country to publish an

opinion "that a positive pressure of gas within an area of softening, or within a definite cavity already formed, helps to produce excavation", and to infer that this might be a common feature. Pearson thought that secretions within a cavity might cause a valve effect after the pattern of the valve in a positive pressure spontaneous pneumothorax. A high pressure would be caused with cough and straining. Whatever the nature of the valve it was believed that such did occur and gave rise to a positive pressure within a cavity. Writing shortly afterwards (1931), Brooke (14) described a case which simulated a localised spontaneous pneumothorax. Needling gave pressures + 4 + 6 and withdrawal of 100 cc. air gave + 2 - 2. Unfortunately no radiological or pathological evidence was available to support the author's opinion that this was a cavity inflated by a bronchial valve and not a spontaneous pneumothorax. The physical signs and evident pleural thickening as sensed when introducing the needle into the space were suggestive, however, of a cavity rather than a pneumothorax.

In 1932 Coryllos presented his conception of the mechanics of cavities whereby he maintained that check valve inflation and atelectatic shrinkage were processes controlled by the draining bronchus and that these mechanisms formed the basis of cavity behaviour. Although it may be felt that Coryllos subsequently tended to overstate his case to the

point of excluding any other mode of cavity closure than occlusion of the bronchus, a great deal of credit is due to him for introducing in a characteristically forthright manner principles of cavity mechanics which are coming to be accepted more and more widely. Eloesser has also come to be regarded as one of the pioneer exponents of the valve mechanics of cavity inflation. But Eloesser gives the impression of having lost his way: the reason for this probably lay in his failure to recognise that complete closure of the bronchus results in cavity closure rather than in complications. Although at first Pinner did not recognise bronchial occlusions as an important means of cavity closure, subsequently this authority came to give a prominent place to this factor in his conception of cavity healing. Pinner, it is felt, ultimately came to uphold a more balanced, and therefore more satisfactory, belief with regard to cavity healing than was the case with Coryllos, who rejected the possibility of the cavity walls playing an active role themselves. In this county as early as 1933, Morland (45) in a very excellent exposition on the formation and treatment of cavities expressed the opinion that it was probable that destruction of tissue and inflation were involved in cavity formation. Agreement may not be felt with all his views at that

time regarding treatment but his remarks concerning cavity formation are very advanced.

Writing on the subject of cavities in 1939, Coryllos and Ornstein declared that nothing new seemed to have been added to the knowledge of cavities during the past ten years, since the general opinion was expressed at the Annual Convention of the German Tuberculosis Society in June 1927, regarding cavitation, that many questions remained unanswered especially with reference to the causes and mechanism of the spontaneous closure of cavities. Now, ten years later again, it still has to be admitted that a great deal yet remains to be added or established regarding the knowledge of the factors governing the behaviour of cavities. Writing more recently (1944) on the subject of tension cavities, Rafferty concluded that none of the theories of the mechanics governing such cavities is entirely satisfactory.

In approaching the subject of persistent cavities as a study for this thesis, it has been felt that a true conception of the mechanism governing the behaviour of cavities can only be reached when an exhaustive survey has been made of all the possible factors which come into play. Only when these factors are regarded in a balanced proportion can a truer conclusion be reached regarding the root causes

underlying the occasions when surgical means fail to close cavities. There are no prospects that with the advent of streptomycin, or other more effective antibiotics, cavitation will cease to be a problem in pulmonary tuberculosis.

The type of persistent cavity which has been studied has been the one frequently to be found in the chronic or arrested case. The active cavity with progressive necrosis within its walls has not been included for the purpose of this study.

The course of pulmonary tuberculosis is long and hard in most cases at the best of times. To this may be added the ordeal of a series of operations, each one resulting in discomfort and disappointment to the patient, months being added to months, leading into years, whilst the cavity persists as an ever-present source of danger to himself and a barrier from his family and society. All the knowledge and experience which can be brought to bear, in order that the quickest and most effective means can be employed early in the treatment of cavities, will result in that much less unnecessary suffering being imposed upon the patient. And what is the purpose of all the treatment of the physician if it is not to alleviate suffering ?

S E C T I O N I.

APPLIED PHYSIOLOGY

The lung consists of the bronchi with their ramifications which end blindly in the pulmonary air sacs. The whole system is closely invested by visceral pleura and contained within the thorax, which is lined by parietal pleura.

The bronchi end as terminal bronchioles. From these latter there branch off a number of respiratory bronchioles which in turn lead into alveolar ducts, then into the alveolar sacs or air sacs of the lung. The bronchi, as far as the terminal bronchioles, act as a conduit system by which air is conveyed to and from the respiratory portion of the lung - namely the respiratory bronchioles and their air sacs, which together form a primary lobule (Best and Taylor (8)). Miller (42) does not include the respiratory bronchiole in the primary lobule.

The bronchi, like the trachea, are lined by a mucous membrane composed of ciliated columnar epithelium resting on a basement membrane; external to which are numerous longitudinal elastic fibres. Outside again are to be found plain muscle fibres which encircle the bronchi as an interlacing network. External to this again is a loose fibrous tissue layer containing mucous glands and, in the larger and medium-sized bronchi, plates of cartilage.

As the bronchioles are followed into the primary lobules, and function changes from that of air conduction to that of respiratory exchange of gases between the contained air and the venous blood of the lung capillaries, various changes take place in the histology of the tissues described. The ciliated columnar epithelium gives place to non-ciliated cubical epithelium as the respiratory bronchioles are approached. The walls of the air sacs are lined by a single layer of flattened epithelial cells supported by delicate connective tissue in which are elastic fibres. Muscle fibres are to be found on the walls of the respiratory bronchioles as far as the distal end of the alveolar ducts, where they form a sphyncter around the openings into the air sacs. According to Vallentin (70), Dykstra has demonstrated a highly developed muscular system within the lung, which includes the alveolar walls and interstitial tissue. Such a finding is denied by Miller (42), however, on a basis of faulty technique. The proportion of muscle tissue in the bronchioles is considerably greater than that found in the rest of the bronchial tree. Comparing the changes in thickness of the muscle with the changes in diameter of differ-:ent types of bronchi, Miller concludes that in a bronchiolus of 1 mm. diameter the muscle bands are five times as strong as in a bronchus in which the diameter is 10 mm. The amount of cartilage in the bronchial wall, on the other hand, becomes less; and at a diameter of less than

1 mm. (Cawdray), or 0·6 - 0·7 mm. (Miller), the cartilage disappears from the bronchial wall. Thus it will be seen that as the bronchi become smaller, there is less cartilage to protect their patency, and an increasing amount of muscle tissue.

The blood supply (Miller) to the bronchial tree, as far as the terminal bronchioles, is from the bronchial arteries - branches of the thoracic aorta. These form a capillary network within the wall of the bronchus, and connect with a rich venous network which drains into the pulmonary vein, except in the region about the hilum where the venous return is into the azygos, hemiazygos, or one of the intercostal veins. The air sacs and respiratory bronchioles are supplied by the capillaries of the pulmonary artery, and the venous return is by way of the pulmonary vein.

Lymphatics are found in the walls of the bronchi, and accompanying the branches of the pulmonary artery and the pulmonary veins. In the larger bronchi there are two plexuses of lymphatics, one being found inside and the other outside the cartilage. In the smaller bronchi there is only one plexus of lymphatics. Miller describes the pleura as being supplied by a single plexus of lymphatics arranged in irregular polyhedral rings. All the lymphatics drain into lymph nodes at the hilum. No lymphatics are to be found in the walls of the air sacs.

The bronchioles are supplied by excitor, or broncho-constrictor, nerve fibres derived from the vagus; and inhibitor, or broncho-dilator, fibres from the sympathetic nervous system. Pilocarpine, histamine and certain foreign proteins capable of producing anaphylactic reactions, have a constrictor effect upon the muscles of the bronchioli. Adrenaline, ephedrine and atropine cause dilatation.

Applying the histology of the bronchial tree to its physiological action in the living subject, it is seen to be composed of strong walls the patency of which is safeguarded by rings or disjointed plates of cartilage. At the same time the bronchus is capable of longitudinal stretching and recoil by virtue of its elastic tissue, whilst its lumen undergoes inspiratory dilatation and expiratory contraction. Best and Taylor, and Rafferty (55) maintain that these bronchial movements are purely passive, responding to the changes in volume of the thorax. Pinner (53) partly agrees with this, but maintains that shortening and narrowing of the bronchus on expiration can only be an active process promoted by contraction of the bronchial musculature. In the smaller elements of the bronchial tree, and where they join the primary lobules, the muscle tissue becomes particu-:larly strong, and, being no longer checked by cartilage, can, under provocation, produce complete spasmodic occlusion of the lumen. The walls have a rich blood supply which renders them susceptible to haemorrhage

and rapid vascular congestive changes. The nature of the blood supply, in conjunction with the lymphatics, facilitates embolic spread of infection within the bronchial walls, or to other parts of the lung and the other organs of the body. At the same time, the mucous glands provide a secretion which, in company with any foreign matter inhaled or extruded into the lumen, is propelled towards the upper respiratory tract by the action of the ciliated epithelium.

The bronchi serve a dual function in the physiology of respiration. It is mainly due to their elasticity, perhaps aided by contraction of muscle fibres, that the expanded lung recoils in quiet expiration. Normal expiration, as opposed to forced expiration, must chiefly be regarded as a returning movement of a resilient organ to its former state after having been expanded. Secondly, the bronchi have already been described as the air conduit system. To fulfil this latter purpose it is essential that the airway be kept free. This is maintained not only by the cartilaginous reinforcement of the medium-sized and larger bronchi, but by various movements of which the bronchial walls are capable.

The bronchi dilate and elongate with inspiration, and contract and shorten with expiration. This latter movement produces an expiratory thrust by the bronchial walls upon the contained secretions, propelling them towards the larger air passages. Best and Taylor describe peristaltic movements of the bronchioli which propel foreign material towards the larger bronchi.

Pinner (53) considers the existence of such movements to be unproven. In addition there is the propelling mechanism of the ciliated epithilium.

Referring to the defensive drainage mechanism of the bronchi, Jackson (33) wrote, "One of the greatest medical mistakes of all history was the ther-:apeutic antagonism to cough." Not only does the blast of air on coughing sweep the clogging secretions towards the larger bronchi, but, as Jackson points out, as a result of bronchoscopic inspection, in coughing it is the compression of the lung, or tussive squeeze, which is one of the most important mechanisms of pulmonary drainage. In the smaller bronchioles and air spaces, the tussive squeeze is particularly important as there may be little, or no, air to get behind the secretions to force them towards the upper respiratory tract. Nevertheless it would be unwise to overlook the harmful effect which may be caused by a dry, exhausting cough, such as is encountered particularly in pulmonary tuberculosis. On these occasions the amount of secretions to be got rid of is small in comparison with the irritation being produced in the bronchi.

The expansion and contraction of the lung during respiration, with the corresponding movements of the bronchial tree as described above, are brought about by movements of expansion and contraction of the surrounding walls of the thorax. The thorax is a closed space, lined by the parietal pleura; the lung surface is covered with the visceral pleura: between

these two surfaces is the pleural space, - a potential space only.

As the walls of the thorax expand outwards, the negative intrapleural pressure is increased, and the surface of the lung is drawn out after them. This is made possible by outside air being drawn into the lung via the respiratory tract: and the act of inspiration occurs. As the walls of the thorax contract down again, the negative intrapleural pull is reduced once more and the lung recoils by virtue of its elasticity, - which is mainly that of the bronchi, air being driven out once more in the process of expiration.

The respiratory movements of the lung are not uniform throughout its substance, however. Certain regions expand more than others; and the direction and amount of movement of individual sections of the lung differ from those of other sections. There is no uniform eccentric expansion and concentric contraction as is the case with an inflated balloon.

Three zones of differing distensibility of the lung have been described by Keith (Best and Taylor (8)): (i) a non-expansile root zone; (ii) an intermediate zone; (iii) an outer or subpleural zone, from 1" to 1¼" deep, of maximum distensibility. But all the portions of lung lying within each of these zones are not equally distended upon inspiration. If this were so, the apex, which possesses the greatest relative amount of subpleural lung, would be the most expansile portion; which is not the case, but rather the reverse. The ex-

-planation lies in the second factor, the unequal movement - as distinguished from expansion - of different sections of the lung.

The unequal movement of the lung is due to the fact that the anatomical structures which constitute the walls of the thorax, and which are responsible for the expansion and contraction of the lung, have different degrees and directions of movement.

The diaphragm, especially in its posterior half, undergoes the greatest range of movement of the walls of the thorax. The ribs move progressively less from the tenth rib towards the first, which hardly moves at all. The vertebral column, which takes part in the formation of the postero-medial wall of the thorax, is immobile. In cases where the intrapleural pressures are unequal in the two hemithoraces, the mediastinum is generally capable of being drawn across towards the side with the greater negative pressure. The diaphragm acts in a piston-like manner. It plays the major role in the movements of respiration. The ribs move with a slight upward and outward swinging movement from their former obliquely downward position. The movement has been likened to the raising of a bucket handle.

The effect upon the lung of these combined movements of the walls of the thorax can best be understood by reference to figure 1. It will be noted that the general movement of the lung on inspiration is obliquely downwards and forwards, away from the immobile walls of the thorax, which are the posterior

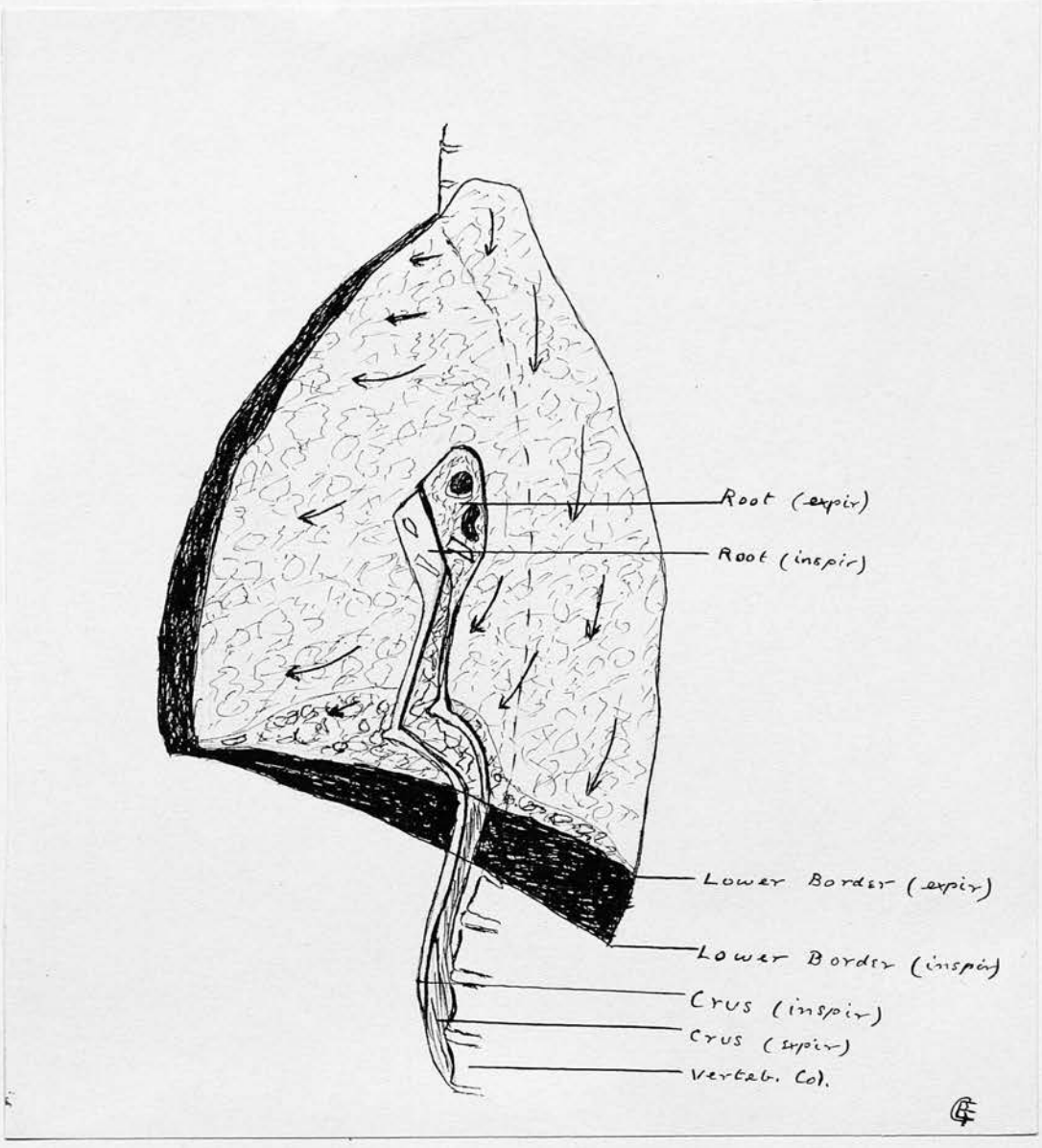


Diagram to show the respiratory movements of the lung. The arrows indicate the direction of the inspiratory movement of the various parts of the lung.

Figure 1.

wall and extreme apex: whilst the main movement of the posterior part of the lung is vertically downwards in the direction of the descending diaphragm. The regions of the lung which lie close to the immobile portions of the thoracic wall - such as the dorsal surface of the apex, the posterior portion in contact with the vertebral column, and the mediastinal part of the lung - can only move indirectly, by the withdrawal of the adjacent lung substance. The apex depends upon the descent and forward motion of the lung root for its respiratory expansion. Thus, to take examples: the subpleural zones at the base and lower anterior portion of the lung undergo the maximum movement and distension; the root zone moves appreciably but distends a minimal amount; whilst the subpleural region of the posterior apex undergoes no movement but a small amount of distension.

S E C T I O N II.

Introducing

THE PHYSICS OF THE INTRATHORACIC PRESSURES.
(Their Mechanics, and their Effects upon the Tissues of the Lung.)

In order to understand the mechanics of cavity formation and behaviour it is necessary to examine the intrapulmonary forces which are at play - that is to say, the nature of the stress and strain imposed upon a given area within the lung during respiratory movements, or under the influence of coughing or straining. It is only by such a means that the behaviour of such an area can be anticipated after its tissues have been disrupted and destroyed, or weakened by necrosis.

The intrathoracic pressures fall into two categories: (1) intrapleural; (2) intrapulmonary. The intrapulmonary pressures are entirely dependant upon the intrapleural pressures; although other mechanical agents such as occlusion of the bronchus, as will be shown later, can be responsible for affecting both these pressures.

INTRAPLEURAL PRESSURE.

Before birth the lung is airless. At birth, due to the expanding action of the walls of the thorax, air is drawn into the air spaces of the lung - and the

connecting the trachea of a cadaver to a manometer, and puncturing the chest wall, thus allowing the lung to collapse, equalising the intrapleural and intrapulmonary pressures. The negative intrapleural pressure increases on inspiration and decreases on expiration - the normal range on quiet respiration being -2.5 to -6 mm. Hg.(8): Pinner (53) gives the range as -2 to -7 cm. water. The pressure remains subatmospheric throughout both phases. With forced respiratory movements against a closed glottis, however, pressures may reach -40 mm. Hg on inspiration, and +50 mm. Hg on expiration. Rapid fluctuations of the intrapleural pressure between extremes such as these, must be kept in mind when considering the effect upon a cavity of a severe bout of coughing.

The negative intrapleural pressure, or tension, is an outwardly expanding force which exerts its influence throughout the lung, drawing the branches of the bronchial tree, and the intervening air spaces, outwards like rays from the hilum. Best & Taylor point out that it is the elongation of these rays, rather than any widening of the spaces between them, which is of importance in permitting expansion of the pulmonary tissue. This force, derived from the intrapleural tension, should only be regarded as acting upon and through the tissues, and not upon the gases contained between those tissues. The tissues

surrounding a disrupted area of lung will be drawn apart by the opposing forces of the negative intrapleural pressure and the retractility of the lung.

There are certain conditions which increase the negative intrapleural pressure. Atelectasis, especially if it involves a whole lobe of a lung, will make the intrapleural pressure considerably more negative on account of the shrunken region drawing upon its surrounds in the process of shrinking: the same applies to pulmonary fibrosis. If the atelectasis or fibrosis is in the contralateral lung the intrapleural pressure is lowered by the mediastinum being drawn across towards the side with the shrunken parenchyma. Under conditions such as these the intrapleural pressure may fall from the normal -6 mm. Hg on inspiration to as low a pressure as -30 mm. Hg.

INTRAPULMONARY PRESSURE.

The greater part of the lung in the living person is composed of air spaces which, being in free communication with the atmosphere, contain gases at atmospheric pressure. Whilst the intrapleural pressure acts as a referred tensile force upon the lung tissue and is generally subatmospheric, the intrapulmonary pressure exists in the gases contained within the air spaces of the lung and approximates to

atmospheric pressure. A needle introduced into the air spaces of the lung - alveoli or bronchi - and connected with a manometer, will register oscillations of pressure in the region of -1 +1 mm. Hg. Best & Taylor give the range of pressures within the air sacs during quiet respiration as slightly less than -2 +3mm. Hg. A prolonged, forced inspiration will create a pressure of -40 to -50 mm.Hg; whilst coughing or straining against a closed glottis will give a pressure of +10 to +40 mm.Hg.

As Best & Taylor point out, the intrapulmonary pressures are considerably increased under conditions of obstruction to the respiratory passages; they can, in fact, be regarded, under such circumstances, as being a measure of the degree of obstruction prevailing. The normal intrapulmonary fluctuations of pressure are a measure of the obstruction which occurs naturally to the passage of air through the bronchial tree on its way to and from the alveoli. The changes in pressure on compressing and releasing a rubber syringe bulb, for instance, would be almost negligible if the bulb had a wide orifice. If the bulb is connected with a narrow glass nozzle, the difference now prevailing in the internal pressures of the bulb will be detectable by the fingers during compression: the pressures will rise and fall dramatically as the bulb is compressed and released.

In the case of the air spaces of the lungs, as in the case of the rubber bulb, air passes in and out of the enclosed space during respiration in its effort to equalise the pressures within and without, having to circumvent whatever obstruction there might be during its passage.

It will thus be seen that the range of fluctuations of the intrapleural and intrapulmonary pressures is much the same; being approximately through 4 mm. Hg on quiet breathing, and rising to as much as 80-90 mm.Hg with straining, such as violent coughing. The notable difference is that whereas the intrapulmonary pressures range on either side of the zero mark in quiet breathing, the intrapleural pressures, under similar conditions, are always sub-atmospheric. The intrapleural pressures may be regarded as a measure of the extrabronchial and extra-alveolar forces, whilst the intrapulmonary pressures are a measure of the intrabronchial and intra-alveolar forces. Furthermore, as has already been shown, the one is a pressure which has been transferred from the pleural space to the tissues of the lung, whilst the other is a gaseous pressure confined to the gases enclosed within the lung.

APPLICATION OF THE INTRATHORACIC PRESSURES TO CAVITY FORMATION AND BEHAVIOUR.

If there is disruption of lung tissue and the area has free access to the atmosphere through the bronchial tree, a hole, or cavity, will form, the walls of which will be composed of the adjacent lung tissue. The inside of the cavity will contain gases at atmospheric pressure, but the walls will be subject to a subatmospheric tension distributed uniformly around them, pulling the lung parenchyma radially outwards and being opposed by the elasticity of the lung tissue. It is for this reason that most cavities tend to assume a spherical shape.

Thus the mechanical forces which are responsible for cavity formation and behaviour must be regarded from two aspects - the extracavitary forces acting upon the walls of the cavity pulling them outwards, and the intracavitary forces which promote internal distension or contraction. These two principles must always be thought of in conjunction when considering cavity behaviour. It is an entire misconception of the machanics of cavitation to think vaguely in terms of pulmonary elasticity and to disregard the pressures of the gaseous contents of the cavity. The dynamics of pulmonary cavitation cannot be reduced to the simple principles of the spider's web.

Such a principle might account for the shape of a cavity but not for its behaviour. Reinders, believing that the retractility of the lung round about caused a cavity to be pulled into the shape of a sphere, illustrated this by burning a hole in a piece of stretched rubber sheeting. Upon increasing the size of the hole irregularly, he demonstrated that it still maintained its circular shape. But it must be realised that a two-dimensional circle freely suspended in the air, is not to be compared with a three-dimensional sphere suspended within a closed container and only communicating with the atmosphere from inside the sphere.

Eloesser (24) describes a spherical cavity as the product of the combination of (1) interruption of the unyielding framework (presumably the bronchi) which anchors the resilient mass of lung to its root, (2) subjecting the resilient lung either to internal air pressure or external suction (pull of the chest wall) and (3) allowing air to enter the site of the rupture. This conception Eloesser partly bases upon the principle illustrated by Reinders, which he augments to form a true conception of cavity behaviour. The only objection to Eloesser's description is that he specifies interruption of the "unyielding" framework. Disruption of one or more primary lobules will give the same spherical cavity on a small scale.

EXTRACAVITARY FORCES.

Certain authorities seek to explain the mechanism of cavity formation and behaviour almost exclusively on a basis of the extracavitary forces, paying little, if any, regard to the intracavitary forces.

Andrus (3) finding no change in the size of cavities from full inspiration to full expiration, concluded from this that a cavity is governed by factors coming into play continuously for hours or days and not by gas pressure relationships inside and outside (pleural space) the cavity. Pride of place is given to "the elastic pull of the lung", to which the cavity owes its patency, enlargement and spheroidal form. If this pull is removed the air chambers collapse by reason of concentric elasticity. The phenomena of cavity fluctuations in size he ascribes to mechanismsof atelectasis: this will be discussed later. The elastic pull of the lung is so directed as to dilate into a spherical hole "the margins of a focus of discontinuity that may arise from destruction", and he adds that cavities are more likely "traumatic artefacts by nature resulting from the dilatation of destructive tears", rather than holes resulting from excavation of tissue.

Rafferty (55) criticizes the experiment Andrus carried out and used to substantiate his theory. Having punctured a hole in the lung of a cadaver, and having tied up the pleural opening, Andrus inflated the lung and noted the appearance of a hole at the site of incision within the lung. This he considered demonstrated that a cavity is caused by the elastic pull of the lung. As Rafferty points out, the experiment "amounts to little more than blowing up a cavity". It is probable that under such experimental conditions the air was trapped within the artificial cavity, partly owing to collapse of the inert bronchioles which would occur with the collapse of the lung upon opening the thorax thus abolishing the negative intrapleural pressure. It would be interesting to know what explanation Andrus would give for the necessity of admitting air into the incised area of the lung before the cavity appeared. Evidently he does not believe that the elastic pull of the lung is capable of separating the walls of a cavity in the face of an incipient vacuum; and yet the admission of air into a cavity forms no essential part of his views on the basic principles of cavity formation. It is apparently taken for granted that the admission of air is necessary.

Andrus is correct when he recognises the

"elastic pull" of the lung as a dilating force in cavity dynamics, but not when he relegates to an unimportant place the relationship between the intra- and extracavitary gaseous pressures.

Coryllos (19)(29) draws attention to, only to condemn, certain theories of cavity behaviour based upon changes reported to take place in the elasticity of the diseased parenchyma of the lung. Parodi, Dumarest and Lefevre, and Van Allen tried to explain the changes in size and closure of cavities on the grounds that there were two kinds of elasticity - one of retraction and the other of expansion. These, they maintained, were capable of independant action. In the tuberculous lung the elasticity of retraction increased over the elasticity of expansion, and cavity closure resulted. Parodi subsequently appeared to abandon this view in favour of another, which Coryllos found equally unsatisfactory. Van Allen, whose views differed perhaps slightly from those of the others, believed that cavity closure was due to increased elasticity of the diseased parenchyma caused by thickening of the interalveolar septa. Coryllos points out that the increase of elastic strength of a tissue can only be brought about by an increase of the elastic elements and that in tuberculosis the elastic component is replaced by unelastic fibrous

tissue, thus decreasing its elasticity. "I do not believe", wrote Coryllos, "that the living lung can be considered merely as an elastic organ submitted exclusively to the mechanical laws of elasticity; it is obvious that the lung of a living patient is a living organ, the physiological functions of which are far more important than its elastic properties."

It is to be concluded that the extracavitary force may cause the development, enlargement and spheroidal shape of a cavity - but provided that the state of the intracavitary gas pressures does not prevent this. These two forces must always be regarded in conjunction, either balanced the one against the other or working in the same direction.

That strong positive or negative intracavitary forces may develop will be shown later. Extracavitary forces may be even stronger. Adhesions of the lung to the chest wall can prevent a cavity from closing although the internal pressure is strongly negative on account of a closed bronchus.

Pottenger (54) describes how a cavity may be prevented from shrinking by certain interfering mechanical factors. These are extracavitary forces contributed by such conditions as pleural adhesions, a fixed diaphragm or mediastinum, dense fibrous tissue or a rigid chest wall. Pottenger points out that

normally the amount of lung tissue present is only just sufficient to fill the thorax: any loss of tissue as a result of disease requires compensation. The above mentioned factors interfere with the giving of this compensation.

As is pointed out by Andrus, even when collapsed by a pneumothorax the lung is still stretched in a state of elastic tension because the intrapleural pressure remains negative. Under these conditions the lung volume shrinks and the cavity volume may shrink with it; but the cavity will still be held patent by the negative intrapleural tension - this prevails until the lung is atelectatic.

Apart from such emergencies as a traumatic or spontaneous positive pressure pneumothorax, a positive extracavitary force is usually only encountered locally within the lung. It may be produced in this way by a positive pressure artificial pneumothorax, or again, in the vicinity of a cavity with a rigid wall or of a cavity distended by a positive internal pressure which is withstanding the efforts of the surrounding lung tissue to shrink in response to collapse processes.

INTRACAVITARY FORCES.

The intracavitary forces, which are the intracavitary gaseous pressures, may be divided into two categories: (1) static forces, which are the constant or mean pressure states within the cavity, and (2) dynamic forces, or the fluctuating changes of the intracavitary gaseous pressures.

By referring to the static and the dynamic intracavitary pressures, one is merely describing two features of one and the same force: the static pressure is the mean of the dynamic pressures.

The static pressure within a cavity may be greater than, equal to, or less than the atmospheric pressure. It is a force acting continuously on the cavity walls from within. This force will, in the long run, determine whether a cavity is going to remain distended or whether it will be subject to a continuous force of contraction tending to close it. If, on the other hand, the cavity contains gases at mean atmospheric pressure, forces other than intracavitary ones will be responsible for its subsequent behaviour: the extracavitary forces or scar tissue retraction within the cavity walls will dominate the issue.

(1) Static intracavitary forces.

It has already been shown that the mean intrapleural pressure is subatmospheric, whilst the mean intrapulmonary pressure is atmospheric. The result is that the intracavitary pressure is greater than the extracavitary pressure to the extent of a mean of 4 mm. Hg: the cavity is patent and spherical in shape in consequence.

But all the pressures which have been considered hitherto have been relative pressures - that is to say, relative to the atmospheric pressure. The whole body, within and without, is subject to the weight of the atmosphere, which is 15 lb. per square inch - or equivalent to the weight of a column of mercury 760 mm. high. Such a weight would crush all but the strongest hollow containers: and this would be the effect upon the body were it not that in this case, the pressures operate inside as well as outside. When the intrapleural pressure is described as being on the average -4 mm. Hg, this means 4 mm. Hg below atmospheric pressure, which is in reality 756 mm. Hg outside the cavity walls tending to crush the cavity in, and 760 mm.Hg inside pushing the walls apart. The cavity is patent because the internal pressure is greater than the external. If it were possible to remove the

atmospheric pressure from within a cavity - in other words create a vacuum - the full weight of the atmosphere would be directed upon the walls of the cavity from without: and there is no cavity within any lung which could withstand such a closing force. This would hold true even though the surrounding air sacs were atelectatic as the pressure would be directed through the airless lung tissue.

The tissues of the body and the blood circulating in the walls of the cavity also contain gases at high pressure. The total gaseous pressure of the venous blood circulating in the lungs is 703 mm. Hg, which is 57 mm. Hg below atmospheric pressure.

When two diffusible media, such as the atmosphere and the blood, containing gases are in contact, diffusion of gases takes place from the medium of higher total pressure gases to the medium of lower total pressure gases until equilibrium is attained. When applied to a gas, or mixture of gases, in contact with a liquid this principle is referred to as the "Law of solubility of gases".

There is a second principle however by which gaseous diffusion between the diffusible air spaces of the lung and the circulating blood is governed: this is Dalton's Law or the Law of partial pressures, which states that "the pressure exerted by a gas in a mixture

of gases is equal to the pressure which the same quantity of that gas would exert were no other gases present. It follows that the total pressure of a mixture of gases is equal to the sum of the pressures of its component gases." (Best & Taylor)

By a combination of the principles of both these laws it will be seen that each individual gas of the mixture of gases, and of the liquid, will diffuse from the medium in which the partial pressure of that gas is higher to the medium in which the partial pressure of that particular gas is lower. The final product will be equilibrium of the total pressures in each medium.

From this it follows that were a state approaching vacuum created within a cavity the gases in the venous blood would diffuse into the cavity space. On the other hand, when the venous blood is exposed to the atmospheric air, as in the alveoli, diffusion of oxygen will take place from the air into the blood because the partial pressure of atmospheric oxygen is greater than the partial pressure of the oxygen in the venous blood. Carbon dioxide, on the other hand, will diffuse from the venous blood into the alveolar air because the partial pressure of carbon dioxide in venous blood is greater than that in atmospheric air. There will thus be a mutual re-adjustment of gaseous pressures within and between

these two media, the venous blood and the atmospheric air, until equilibrium is attained and their total pressures are the same. This principle has been demonstrated experimentally in the case of cavities by Coryllos (see page 73).

If it were possible for a state to be created within a cavity such that the total pressure had been lowered and maintained at -57 mm. Hg, under such conditions no diffusion of gases would take place in either direction, provided that the partial pressures of the individual gases within the venous blood and within the cavity had equalised. From this it would be expected that the greatest negative pressure that can be maintained for any length of time within a closed cavity, in life - other than by artificial means - is -57 mm.Hg. Such an internal force, if it existed, would exert a very strong influence towards closure of the cavity. According to Coryllos (20), such an intracavitary pressure has been demonstrated by Henderson & Henderson, who recorded a pressure of -56 mm. Hg within a cavity where the draining bronchus was closed. Under these conditions the atmospheric air is cut off from the interior of the cavity and the entrapped gases are diffused into the blood circulating in the cavity walls.

In the resilient lung the diffusion of gases does not stop with the attainment of a pressure of -57 mm.Hg. If the cavity walls are resilient the

physics governing atelectasis comes into force. The principles of atelectasis are governed by the same two laws - Dalton's Law and the Law of solubility of gases.

"When a bronchus or bronchiole in a previously distended lung is obstructed the imprisoned air soon becomes absorbed from the affected alveoli. Collapse of the air sacs cannot take place until this has occurred. Absorption is brought about in the following way, as pointed out by Henderson. The air in the isolated alveoli has a total pressure of 760 mm. Hg. The partial pressures are in round numbers, O_2, 100 mm.; N_2, 570 mm.; CO_2, 40 mm. and aqueous vapour, 47 mm. In the venous blood the total pressure is 703 mm., the nitrogen and aqueous vapour being the same as that of the alveolar air, but the partial pressure of oxygen is only 40 mm. and of carbon dioxide 46 mm. An interchange of the latter gases therefore occurs between the alveolar air and the venous blood. It might be thought that the imprisoned air would then be in equilibrium with the blood and no further absorption occur. But the alveolar air loses more oxygen than it gains carbon dioxide whereupon its volume is reduced. The atmosphere acting upon the body surface and through the yielding soft tissues compresses the air so as to maintain its total pressure practically constant at 760 mm. As a result of the absorption of oxygen the percentage

and consequently the partial pressure of carbon dioxide and of nitrogen are increased. These gases now diffuse into the blood. The volume of the air is further reduced thereby but its total pressure still remains unaltered. The percentage and therefore the partial pressure of oxygen rises and more of this gas passes into the blood. The process continues in this manner until no air remains, and the walls of the original space are ultimately approximated by the pressure of the atmosphere." (Best & Taylor p.370)

Eloesser, in 1932, realised this principle with reference to atelectasis of the alveoli when there is total block of a bronchus: but it was Coryllos who, at about the same time, realised that the principle of atelectasis might be applied to cavity closure. Eloesser in fact subsequently stoutly opposed Coryllos and maintained that "total permanent obstruction (of a bronchus), if there is such a thing, is also a factor that causes cavities to resist attempts at closure." The impression gained from reading the views of these two authorities is that whereas Coryllos is definite, and indeed forthright, and founds his arguments upon sound physiological and physical principles, Eloesser is less certain of himself and is groping his way towards an explanation of cavity behaviour. It is this principle of atelectasis applied to cavity closure

which Andrus overlooks or brushes aside when he proclaims that the elastic pull of the lung is of more importance than the relationship of the gaseous pressures inside and outside a cavity.

It cannot be assumed however, that in every case where there is a blocked bronchus the air in the cavity will be absorbed and the cavity closed. It will be noticed in the application of the two gaseous laws to the explanation of the mechanism of atelectasis, that the pre-requisite for further absorption of gases is further compression in order to maintain a constant pressure of 760 mm. Hg. This compression is made possible by the ability of the soft tissues to yield to the surrounding atmospheric pressure. Whilst this is applicable in the case of the alveoli giving rise to alveolar atelectasis, it does not apply in every case to a cavity, especially if that cavity possesses thick connective tissue walls, or walls held apart by strong adhesions. There must arrive a stage during the contraction of such a cavity when further compression cannot take place - at least not for a while, until the connective tissue walls have retracted or the adhesions have stretched or been cut. An old, thick-walled cavity with a closed draining bronchus may persist for a long time, with air trapped inside at a pressure of approximately -57 mm. Hg, waiting until

further tissue retraction of its walls shall have taken place before any more air can be absorbed. On the other hand, a large, thin-walled, resilient cavity with a closed bronchus would close rapidly by a process of atelectasis.

Another dependant factor in cavity closure by the absorption of air, is the ability of the circulating blood to have access to the air within the cavity. Ornstein & Ulmar describe a type of cavity (Type V page 68) with a closed draining bronchus in which no gaseous exchange takes place through its walls because they are thick and avascular. The cavity remains unchanged and resists collapse therapy in consequence. It is perhaps a little difficult to believe that such a cavity could exist, where there was not even one small capillary vessel throughout the whole of its interior stratum which had access to the contained air and which in the process of time would not be capable of absorbing the gases within the cavity. It must be borne in mind, however, that most cavities are lined by necrotic material and secretions. These must inevitably act as a barrier between the enclosed air and the underlying blood vessels in the cavity walls, slowing down, to a greater or lesser extent, the rate of air absorption.

Just as a continuous negative pressure may be established within a cavity by means of closure of the

draining bronchus, causing a tendency for the cavity to close, so the reverse procedure may also take place. By means of a one-way check valve mechanism within the bronchus, or at the broncho-cavitary junction, air may enter the cavity but be unable to escape thus creating a mean positive intracavitary pressure. Such a cavity, which will be described later, will persist until the valve mechanism ceases to be effective.

It will be seen that the static intracavitary pressures are governed by the state of the draining bronchus. This will be dealt with more fully when considering the bronchial pathology as it affects cavity behaviour.

(2) Dynamic intracavitary forces.

A positive static pressure within a cavity must be regarded as exerting a distending, and therefore traumatising, effect upon the cavity walls. The greater the relative positive pressure within the cavity the greater will be the amount of stretching that is imposed upon its walls. If in addition the walls are kept in a state of constant movement, being alternately stretched and relaxed with respirations or coughing, a cavity will be subject to an additional traumatising influence from dynamic intracavitary forces. Just as rest to the cavity walls is conducive to their healing, so constant movement would tend to

encourage activity of the disease process and delay healing.

The extent of the contraction and expansion of a cavity walls is inversely proportional to the range of the oscillations of the intracavitary pressures. The reason for this apparent paradox is that the cavity expands and contracts with movements of respiration and coughing under the influence of the extracavitary forces, which are in turn operated by the movements of the walls of the thorax. But the amount of this contraction and expansion of the cavity is governed by the degree of patency of the draining bronchus. If the bronchus is widely patent the cavity can expand by admitting atmospheric air. But in admitting air it is at once tending to equalise the pressures within and without the cavity, thereby reducing the range of the oscillations of pressure. If the bronchus is closed no air can be admitted and the cavity can only expand by an increase in volume of the gases entrapped. This process will be governed by Boyle's Law which states that "when the volume of a gas is altered, the temperature remaining constant, the pressure varies inversely". The change in volume of intracavitary gases where the bronchus is closed will be out of all comparison to the change in volume which will take place when air passes in and out of the cavity.

through an open bronchus. A cavity with a closed bronchus therefore, will show little change in size with respiratory movements of the thorax; whereas when the draining bronchus is open the cavity will be expected to show changes in size which correspond to the respiratory movements of the thorax. This hypothesis is confirmed by Coryllos (20) who concluded, as a result of cavernoscopic study of cavities in the living subject, that a cavity will expand and contract with the surrounding lung only if the draining bronchus is open to the atmospheric air.

The intracavitary pressures, on the other hand, show a greater range of oscillations when the bronchus is closed, although the walls are relatively immobile. In this case the cavity will enlarge its volume, and with it the volume of the constant amount of entrapped air, to such a degree as will cause the intracavitary pressure, in accordance with Boyle's law, to remain equal to the extracavitary pressure.

This combined hypothesis is supported by the work of Vineberg and Kunstler (71) who, as a result of needling cavities in 54 cases, came to the conclusion that the greater the oscillation in the manometer the smaller the bronchus draining the cavity; and that when there is a large, patent bronchus there are practically no oscillations, and the intracavitary

pressure is approximately zero.

At this stage it would be well to examine the work of these and other authorities in order to try to assess what happens to the cavity walls, in the way of movement, in actual life. Andrus (3) X-rayed 100 thin-walled cavities in full inspiration and again in full expiration. The cavities were measured and compared, and, except in one case, he detected no change in size. The findings of Eloesser, Vineberg and Knutsler, and Coryllos, who have introduced needles into cavities and recorded the pressures, show that the changes in these intracavitary gas pressures is comparatively small, though dealing with such comparatively small air spaces. The pressures vary greatly in different cavities and, as has already been stated, the experience of authorities points to the size of the lumen of the draining bronchus affecting the range of oscillations. Vineberg and Knutsler, as a result of their experience, gave as a guide to assessing the type of cavity entered three examples of intracavatary pressure readings. Whilst the pressure can be built up within a tension cavity to + 30 upon coughing, the greatest range of pressures they quote is - 8 + 8 on forced breathing - curiously enough in a cavity with an open bronchus where, according to their own views one would be led to expect the smallest range of

oscillations. In a cavity with a completely blocked bronchus the initial pressures may be -10 -7, and on forced breathing -12 -5. Coryllos (19), contrary to what would be expected, found absent oscillations when the bronchus was closed, and very slight oscillations (-1 +1 mm Hg) in cavities with an open bronchus. Eloesser (24)(25) similarly recorded small pressure fluctuations. Clinically therefore, cavities usually show no detectable change in size and comparatively small fluctuations in pressure with respirations.

The reasons for these findings lie in three factors concerned with a cavity and the tissues in which it is situated: (1) the rigidity which is often to be found in different degrees in cavity walls; (2) the spongey nature of the lung tissue; and (3) the narrowing of the lumen and the draining bronchus which is to be found in various degrees, especially at or near the broncho-cavitary junction. Similarly the discrepancies in the clinical findings quoted above can most likely be attributed to causes associated with these factors - allowance having been made for experimental errors. Most cavities which have persisted for any length of time will have developed a certain amount of connective tissue within their walls which will give the cavity the ability to withstand

some of the pressure influences imposed upon it. Secondly the stress and strain from the changing intrapleural pressures is conducted through the elastic, resilient lung tissue. A sudden fall in intrapleural pressure will be conveyed through elastic tissue to the region of the cavity. By the time it gets there much of its violence will have been absorbed by the elasticity of the bronchi and air sacs. Similarly a sudden compression of the lung will be conveyed through a cushion of tissue. This spongey property of the lung has the capacity of absorbing the shocks of such acts as violent coughing and sudden straining. This is why the fluctuations in pressure in a rigid-walled cavity, though the volume of its contained air is small, might not differ much from the pressure variations within a rigid-walled pocket of pleura although the air space in the latter is much greater in comparison: the surrounding tissues (pleura and thoracic wall) in the latter case are more rigid than that of the cavity. A strain that is prolonged will eventually be conveyed through to the area of the cavity: in the case of the elastic wall, it will eventually equal the negative intrapleural pull; but in the case of compression the surrounding spongey tissue will yield place to the cavity walls,

if the cavity is rigid-walled or distended with air, and these latter will not be compressed to the same extent as they would have been had the intervening parenchyma been solid - provided, of course, there was something solid against which the cavity could be compressed.

Thirdly, in most cases there is an element of stenosis of the draining bronchus. This will have a buffering effect on the intracavitary pressures by regulating the passage of air in and out of the cavity. Monaldi (43) referring to this subject states that usually the "outlet bronchus or the total of the bronchial ways which open into the cavity are insufficient to achieve equilibrium between the incoming and outgoing of the air and the modifications of volume due to the respiratory process. As a result there is a constantly oscillating condition with a negative parabola descending in inspiration and a positive parabola rising in expiration". Monaldi refers to the outlet bronchus from a cavity functioning as a narrowed way but, as it will be shown later, this narrowing is increased as a result of endobronchial inflammation, which Manaldi does not mention here.

From this point of view it is pointed out that

a cavity situated in the more expansile subpleural zone is served by a narrow bronchus. Such a cavity is therefore liable to be subjected to a greater degree of regulation by bronchial obstruction than in the case of a cavity situated in the relatively non-expansile root zone.

To recapitulate what has been said: the static pressures within a cavity, except when they are atmospheric or zero, play an important part in cavity behaviour by constituting a constant force directed either towards closure or towards distension of a cavity. When the draining bronchus of a cavity is closed a high negative pressure is likely to result within the cavity owing to absorption of the contained air. This will exert a strong closing influence upon the cavity. If the walls of the cavity are resilient, air absorption will proceed until the cavity is completely closed by a principle of atelectasis. On the other hand, by the formation of a one-way check value an equally high positive pressure can be built up within the cavity, distending it in such a manner as to defy all attempts at closure until the valve has been overcome. The dynamic forces within most cavities play a relatively small role when compared with the static forces. The amplitude of the pressuresand the amplitude of the movements of the

cavity walls are not, as a rule, great enough to cause any significant immediate effect upon either the closure or the maintenance of a cavity. Such forces must, however, undoubtedly have a deterrent effect upon the healing process of the cavity walls, which will influence any long-term process of healing by scar tissue retraction. A high positive static pressure can balloon out a cavity to a large size, the intracavatary pressure increasing without any corresponding change in the extracavitary pressure. When a strong dynamic pressure is produced in a cavity, on the other hand, as for example during a severe bout of coughing, an equally strong extracavitary pressure is produced, both pressures being caused by the compression of the lung by the walls of the thorax. The walls of the cavity being thus supported on the outside, any undue strain is prevented.

So great was the significance Coryllos attached to the state of the intracavitary gas pressures that as early as 1933 he wrote "If this conception is correct the most important part in the closure of a cavity is not played by the wall but by the connecting bronchus. Rigidity and thickness, or elasticity and thinness of the wall will only determine the rapidity of absorption." Subsequently

Coryllos classified cavities solely according to the pressure of their contained gases as determined by the state of the draining bronchus. The gases within a cavity might be under positive pressure, at atmospheric pressure or a third type of cavity may have pressures changing from time to time between these two types. Eloesser stresses the role played by air trapped within a cavity in the maintenance and production of cavities and as constituting a frequent source of their resisting attempts at closure.

EFFECTS OF THE EXTRACAVITARY AND INTRACAVITARY FORCES COMBINED.

Andrus has been criticized for giving undue place to the "elastic pull of the lung" and scant regard for the intracavitary pressures. In a mechanism so complex as cavity formation and behaviour no single factor can be given pride of place if this implies minimising other factors unduly. Cavity behaviour cannot be regarded simply as a mechanical problem. Due regard has also to be given to the pathological factors which play their part.

Monaldi (43) describes the effect which he considers is produced upon cavities as an everyday occurrence by the combination of extracavitary and intracavitary forces. Ordinarily in expiration there is a positive pressure within a cavity brought about by the narrowing of the bronchial exit. In inspiration the cavity is subject to the eccentric "parietal" pull of the lung. Both these factors will tend to dilate the cavity. Meanwhile, during expiration the lung tissue surrounding the cavity is placed between two converging compressing forces, the positive intracavitary pressure and the extracavitary concentric force of the recoiling lung tissue. The result is atelectasis of the pericavitary air sacs which further increases the size of the cavity. The cavity thus increases in size without any effective loss of tissue. The implication of Monaldi's hypothesis is that the

forces which normally act upon a cavity tend towards increasing the size of that cavity rather than helping it to close. The radiological appearance of a ring of atelectasis surrounding a cavity is of common occurrence, especially around tension cavities where such is never absent when situated in aerated lung tissue. Moreover, support for this argument is given from the fact that much of this hypothesis is dependent upon the expiratory phase which normally exceeds the inspiratory phase of respiration. According to Morland (45) microscopic studies of the walls of minute early cavities, carried out by Gloyne, showed compression of the surrounding alveoli suggesting a cavity under pressure.

Eloesser (1941), largely as a result of experiments conducted upon cadavers, concluded that most cavities are in a state of partial inflation, that "even irregular, ragged-walled cavities situated in densely infiltrated, largely airless areas of lung contain air which is under more than atmospheric pressure a considerable part of the time". This Eloesser concludes is brought about by the valve effects created by such agents as bronchial secretions. In so far as Eloesser's opinions are based upon his cadaver experiments, they cannot be accepted. In these experiments Eloesser sought to reproduce the movements of respiration and of cough within a lung, by connecting the

trachea to a pair of bellows and recording intra-cavitary pressures by a needle introduced through the chest wall and connected with a manometer. The inflation of a dead lung with bellows is not the same as air being drawn into a living lung as a result of an increased negative intrapulmonary tension. Furthermore inflation is carried out after the lung has been partially collapsed when the pleural cavity of the cadaver was opened. The experimental method applies an intracavitary pressure only and omits the normal extracavitary forces; and also omits the physiological movements of the bronchi. Eloesser's opinion that the intracavitary pressure is above atmospheric most of the time in patent cavities is more acceptable if regarded as being the views of one who undoubtedly has had considerable experience investigating intracavitary pressures. Up till 1941 Eloesser had needled cavities in over 60 patients under X-ray control, observing the intracavitary changes of pressure as recorded by the manometer under such conditions as sniffing, also injecting and aspirating air, and observing the excretion of methylene blue and lipiodol injected into the cavities. But it is considered that some of the experimental methods employed by Eloesser are unreliable - such as the interpretation of cadaver experiments, and relying too much upon assessing the patency of a draining bronchus by observing the

excretion of dyes from the cavity.

A cavity is frequently dominated by a disproportion of one or other of the forces acting upon it from outside or from within. If the bronchus becomes blocked, even for a short time, the intracavitary pressure may become rapidly negative: a positive pressure check valve might suddenly come into force: or the cavity may be held patent by pleural adhesions. When interference of this sort does not occur, however, it must be concluded from what has gone before, that the combined effect of the intra- and extracavitary forces is normally towards enlargement rather than contraction of the lumen of the cavity. A further effect of these same forces is the pericavitary atelectasis which increases still more the size of the lumen.

Experimental support for these conclusions must inevitably be difficult as it would imply knowing in detail the state of the whole draining bronchial tract.

It will be realised that Eloesser's hypothesis is mainly dependant upon a check valve mechanism. There are, furthermore, other important factors in connection with Monaldi's hypothesis to which reference will be made later (page 54).

S E C T I O N III.

Introducing

THE PATHOLOGY OF THE BRONCHUS AND CAVITY WALL.

Having examined the physics of the forces which act upon the lung, and having assessed the degree to which each will affect a diseased area from the point of view of forming, maintaining or closing a cavity, it is necessary now to examine the subject in the light of the pathological processes which occur in the lung in tuberculosis: to see how far these processes are responsible for bringing about these mechanical effects, and whether the pathology is capable of playing a role on its own in cavity behaviour, apart from the mechanical processes.

THE BRONCHUS

As the state of patency or otherwise of the draining bronchus has been shown to be the dominant factor in controlling the atmospheric pressure changes within the cavity, it is perhaps fitting to deal with the pathology of the bronchus first.

In 1923 Ameuille & Levesque (2) drew attention to the pronounced X-ray appearances of the bronchus draining a cavity. The draining bronchus, which the authors have been able to confirm at post-mortem, appears as two opaque, parallel lines running from the

cavity to the hilum. These changes they ascribed to the bronchial walls being wider and thicker than normal. The increased width they considered to be due to paralysis of bronchial musculature. At the same time the walls undergo tuberculous changes similar to those found in the walls of a cavity and in consequence become thicker.

The appearances described by Ameuille & Levesque are familiar to all tuberculosis clinicians, and are to be seen in the X-rays of the cases presented at the end of this thesis. In connection with tension cavities these appearances have come to be known as "racket cavities" or the "stiel-kavernen" (stem cavities) of German writers.

It is only within recent years that the importance of infection of the tracheo-bronchial tree has been fully recognised. It has attracted considerable attention recently, as reference to the Medical Annual since 1943 will testify. Tuberculosis tracheo-bronchitis has become a study in itself.

When considering the incidence of bronchial affection in pulmonary tuberculosis it will be realised that in one respect the disease frequently originates in the bronchial tree rather than in the air sacs. Miller believes that the transitional zone from the bronchial to the pulmonary arterial systems is a favourite site for tubercles to develop - this is in

the terminal bronchiole as it approaches the respiratory bronchiole. Rich (57) believes that it cannot be said whether the infection begins in the alveoli or the bronchus. As both are so adjacent either must involve the other quickly. It is likely however that aerogenous infection begins in the alveoli. This however does not constitute what is known as tuberculous bronchitis, and it is the more advanced stage of the disease which is at present under consideration, when a cavity has formed and more or less of the draining bronchial tract is involved in the infection. In this respect Rich declares that he has never encountered an instance of isolated caseous bronchitis, and that such, as the origin of phthisis must be regarded as being very rare.

Statistics vary according to the amount of the tracheo-bronchial tract under consideration and the stage of development of the bronchitis.

Edwards (1943) quotes that tracheo-bronchial tuberculosis is said to occur in over 10% of cases of chronic pulmonary tuberculosis. According to Alexander (1) tuberculous bronchitis is quoted as being found in 11% of cases of tuberculous parenchymal disease.

Not unnaturally the incidence of the disease increases as consideration is given to the more distal branches of the bronchial tree where the bronchi are smaller and more delicate in structure, and are more

likely to be involved and incorporated in the disease process.

The pathological studies by Overholt & Wilson (47) of specimens resected at operation showed that "tuberculous bronchitis originates in the small bronchi draining the parenchymal foci, extends towards and may eventually involve the main bronchus. Thus in all cases where involvement of the main bronchus is found, lobar and segmental bronchi have likewise been found to be involved". These authorities found 12 (50%) of 24 lobes removed by lobectomy., and 31 (86%) of 36 lungs removed by pneumonectomy showed involvement of the segmental bronchi. The main bronchus was involved in 20 (56%) of the 36 pneumonectomies. Of the 24 lobes examined following lobectomy, four showed tuberculous involvement of the lobar bronchi. Such a high incidence of bronchial involvement they consider would indicate that the tuberculous involvement of the segmental bronchi is almost always associated with parenchymal tuberculosis.

Shipman & Carr (65) found tuberculous changes in the bronchi draining cavities in 10% of post-mortems on tuberculous lungs. They add however, that it was common for them to find some involvement of the bronchial wall producing partial stenosis.

Auerbach & Green (4) believe that tuberculous bronchitis which will occlude a bronchus is frequent. These authors point out that one cannot regard the normal bronchial structure as running up to the cavity wall. The draining bronchus is almost invariably involved, in the region of the broncho-cavitary junction, in the same tuberculous process as the cavity. How far this extends along the bronchus is of course another matter.

According to Coryllos & Goldberg (29) the draining bronchi always show more or less advanced tuberculosis lesions which vary from allergic oedema of the mucosa to caseation, necrosis or fibrotic changes. The bronchial outlet from the cavity constitutes the most important factor in the evolution of cavities.

According to Rubin (59) caseous bronchitis is estimated to be present in 10% of cases of adult pulmonary tuberculosis. This authority describes the smaller bronchi as becoming involved through incorporation in the tuberculous process. At autopsy ulcers are often seen in the walls of the bronchi leading into tuberculous cavities. Judged by the examination of surgically removed lobes ulcerative caseous disease of the smaller bronchi is more frequent then is generally realised.

Rich states that caseation of the lining and wall of a macroscopic bronchus is common as a secondary feature to infection in the related portion of the lung or to encroachment from an adjacent lymph node. As a primary feature it is rare.

Lemoine and Langeard (38) as a result of bronchoscopic studies believe that the activity or otherwise of a cavity can be estimated by observing the mucosal changes of the draining bronchus. The bronchus reflects, as it were, the state of activity of the cavity. When the pulmonary lesion becomes active and the cavity is about to discharge secretions, the bronchial mucosa becomes red and swollen, and often produces increased local secretions of its own. These changes are typically confined to the bronchi which drain the secretions from the cavity. The authors deduce from this fact that infection of the bronchial mucosa occurs by direct seeding of the tubercle bacilli, as opposed to lymphatic invasion of the bronchial wall.

Samson and co-workers (63), as a result of post-mortem studies of 122 cases similarly came to the conclusion that the predominant mode of mucosal infection was by direct contact with tubercle bacilli from cavities. Apparently about one third of the bronchi, however, showed evidence consistent with mucosal involvement following infection of the

peribronchial lymphatics. Bronchial infection in general appeared to come from a distal source and not from the larynx or trachea. In the case of bronchial ulceration produced by contact infection in this way, Rich points out that one is dealing with massive and long-continued infection in which secondary organisms generally play a part. It may be, Rich adds, that the tuberculous infection only occurs at some site where the protective epithelium has been destroyed by the secondary pyogenic infection, or where a large collection of tubercle bacilli come to rest against a sensitized area of epithelium and cause damage through a hypersensitive reaction. That the bronchi are not easily infected through the lumen he considers is evidenced by the fact that it is strikingly common to find bronchi at a distance from a discharging pulmonary lesion which are free from ulceration and yet large numbers of tubercle bacilli are continually passing through on their way from a cavity to the trachea. Rubin mentions the possibility of haematogenous implantation of tubercle bacilli. Rich considers that whilst this mode of infection is possible it is rare for it to start in the bronchus by this means.

There appears to be fairly general agreement amongst authorities upon the pathological changes which take place in tuberculous bronchitis. The condition

has been studied both bronchoscopically in the living subject and pathologically in the cadaver and in pneumonectomy and lobectomy specimens. The following description is based mainly upon the bronchoscopic observations of Jackson (33) and the bronchoscopic and pathological findings of Samson, Barnwell, Littig and Bugher (63). From what has already been said, it seems probable that tuberculous invasion of the bronchus generally takes place as a result of contact infection from the secretions of the diseased lung parenchyma or especially a cavity; that the nearer the cavity the more likelihood there is of the bronchus being affected - the broncho-cavitary junction commonly being inseparable pathologically from the walls of the cavity. Following invasion the mucosa becomes hyperaemic and swollen with oedema. The process spreads to the submucosa where infiltration may progress beneath an intact but inflamed mucosa. Samson believes that it is common for tuberculous deposits to form in the crypts of the mucous glands. If that is the case, it will be seen that as these lie in the connective tissue external to the muscular layer such infection would be deep within the bronchial wall. The disease might not progress beyond the stage of an inflamed, swollen mucous membrane. On the other hand small tubercles may develop which in turn might be followed by ulceration. At first the ulcers may

be mere pin points of yellow in the mucosa. Ultimately, as necrosis about the ulcer develops, granulation tissue will be formed, and the disease might progress to considerable destruction of the bronchial wall. Should the endobronchial disease progress to the stage of ulceration any healing will be by fibrosis.

Coryllos (19), and Brunn etal(16) refer to allergy as a possible origin of the mucosal hyperaemia and oedema on occasions.

To understand what happens in the living subject, it is necessary to correlate the various pathological changes which might occur within the bronchus with its physiological action..

The oedematous mucosa will narrow the lumen of the bronchus. In the larger bronchi this may be of little consequence from the point of view of causing obstruction. In the bronchioles, on the other hand, obstruction is liable to result. According to Stivelman (68) post-mortem studies indicate that in the majority of instances it is the smaller bronchi that drain cavities. Nevertheless, as Coryllos points out, in the case of cavities which develop to a large size by progressive destruction of tissue, the larger bronchi will become involved in the necrosing process.

Unless the obstruction thus caused is complete throughout the whole respiratory cycle air will be able to pass through the narrowed bronchus on inspiration to the distal parts of the bronchial tree

and the air sacs, but will be unable to escape during expiration owing to the contraction of the bronchial lumen which normally occurs during that phase. In this manner a one-way check valve will be established which will have the effect of distending with a positive air pressure a cavity situated distally. The physiological movement of contraction and expansion of the bronchial lumen is by its very nature so liable to cause such a check valve mechanism whenever that lumen becomes narrowed that such a mechanism might be expected to be fairly common in pulmonary tuberculosis. This is further supported by the apparently high incidence of endobronchial affection, especially at and near the broncho-cavitary junction. If the bronchial lumen is narrowed it will become more narrowed on expiration and less narrowed during inspiration: this would give rise to a greater degree of positive pressure on expiration within a distal cavity than of corresponding negative pressure on inspiration - greater in length of time as well as amount.

As has previously been shown (page 44) Eloesser & Monaldi maintain that there is a natural tendency towards enlargement and partial inflation of cavities. In expressing his views at that time, Monaldi made no mention of the physiological movements of the bronchi or of narrowing of the lumen due to

endobronchial affection, both of which will further enhance the tendency towards cavity inflation. Whether Monaldi had these in mind at the time is not clear. It is evident that Eloesser did for he refers to the valve principles which he described in 1938 (24) which include both these factors.

If the foregoing be correct, it would be concluded that under such conditions there would be emphysema of the air sacs distal to the check valve mechanism and, should cavitation exist within that segment, a positive pressure cavity. This however does not conform with clinical experience. As Pinner points out (53) a positive pressure cavity will be found, but the segmental emphysema is seldom seen.

This at once raises two controversial elements: why is there no emphysema? and secondly, does this mechanism of contraction and expansion of the smaller bronchi actually occur as a general rule in the presence of endobronchial disease? If it does not, then some more localised form of check valve mechanism might be sought as a cause of cavity inflation. The first question raises the problem of the etiology of emphysema: this however will not be entered upon. The second question, being even more pertinent to the subject of persistent cavitation, requires close examination. As however little appears to be known upon the subject its treatment must needs be brief.

If segmental emphysema is not apparent in connection with positive pressure cavities, three possible explanations present themselves, any one of which would satisfy the question up to a point: (1) the valve effect is generally limited to the region of or near the broncho-cavitary junction; (2) the feature is governed by a principle of unequal gaseous diffusion in the air sacs as compared with the cavity; and (3) the condition occurs more often than is realised but is being missed. These explanations will now be examined individually.

(1) The valve effect is limited. Such a hypothesis would appear reasonable in view of the origin of the bronchial involvement - the cavity. As Auerbach and Green point out (page 49) the bronchus in proximity to the broncho-cavitary junction is almost invariably involved. This also accords with the post-mortem findings of Loesch (40) (figure II). The nearer to the source of infection, the greater will be the degree of bronchial involvement. It seems likely that the maximum valve effect is created near the cavity, and that this must especially be the case when a strong positive pressure is created within the cavity.

But the fact must be accepted that generally more of the draining bronchus is involved than a short section near the cavity. The experience of Lemoine

and Langeard (page 50) indicative of this fact, is also supported by the clinical findings in the cases presented with this thesis where all three of the cases examined by bronchoscopy showed inflammation of major bronchi. This being the case, a more widespread form of check valve must be produced which will involve a greater portion of the lung than the immediate cavity area. As will be shown later, a valve of this type does not create a strong positive pressure unless stenosis is so great that air can only be admitted distally during a forced inspiration, as in coughing. A high negative intrapulmonary pressure leaves a high positive pressure beyond the check valve. Furthermore, it does not require a strong positive pressure to keep a cavity inflated and patent.

Thus far the answer to the question why emphysema is not found around a positive pressure cavity is that in most cases the area involved is not large; and where the air sacs of the lung are involved in a check valve mechanism, a strong enough positive intra-alveolar pressure is not produced to cause any appreciable degree of inflation. The explanation, however, partly lies in the next feature to be considered - the alveolar air is rapidly absorbed.

(2) Gases diffuse more rapidly through the walls of the air sacs than through the walls of a cavity. This is a reasonable assumption. Gases

contained within a cavity which is lined with secretions and possesses a relatively thick wall will be absorbed more slowly than gases contained within resilient, thin-walled air sacs. If it is assumed, as suggested by the experimental evidence of Brunn et al (16), that air is admitted beyond the valve only at infrequent intervals, there will be plenty of opportunity for air to be absorbed within the air sacs. If, on the other hand, air is admitted regularly with inspirations, the nature of the valve must be such that the positive pressure created cannot be great.

Furthermore, not only will gases be diffused into the blood stream from the alveoli, but there is evidence to suggest that interalveolar or interlobular canaliculi exist: or as Josselyn's studies suggest (8) the alveolar lining may be a discontinuous membrane; Cowdray (21) describes "alveolar pores" of a similar nature. Such, if present, would provide an additional means of decompression either through channels or by way of rapid diffusion. No such systems can be conceived as existing in the zone of atelectasis and pathological changes which immediately surround a cavity.

The principle of unequal gaseous diffusion would explain the absence of emphysema when, as must at times occur, a valve mechanism capable of creating

a strong positive pressure is formed at some distance from the cavity along the bronchial tract.

This process may eventually lead to atelectasis rather than emphysema, around a cavity. This is sometimes seen with an artificial pneumothorax following adhesion section. Lobar atelectasis occurs round about an inflated cavity. Here there has been a pronounced degree of bronchial occlusion: the balance has been swung so far in the direction of limiting the amount of air which can pass the valve that only the cavity remains inflated by virtue of its slower absorption of air. Pinner (53) believes that peripheral to a bronchial stenosis under-aeration and shrinkage of the parenchyma are more frequent than localised emphysema and attributes this to inflammatory processes plus stagnant secretions causing clogging of the bronchioles and fibrous shrinkage.

Eloesser (23) was puzzled by a similar and perhaps very relevant phenomenon. He noticed that when there was stenosis of the larger bronchi, the depending smaller bronchi became dilated, sometimes forming tubular or bottle-shaped endings. This involved the bronchial system only: there was no emphysema. Eloesser compares this to blockage of a ureter, which results in hydronephrosis but not dilatation of the tubules; and to constriction of the ampulla of Vater leading to dilatation of the

common bile duct but not so regularly of the finer biliary tubules. A similarity of underlying principles suggests itself - of differences of gaseous diffusion in the case of alveoli and a cavity, and the osmotic absorption of fluids with their dissolved salts and suspended colloids which does not take place at all in the kidney pelvis or the common bile duct. Eloesser was further puzzled by finding that when there was constriction of the bronchioles the result was irregular emphysema. This might be accepted as further suggestive evidence of inter-alveolar communication.

(3) One must speculate upon the possibility of segmental emphysema occurring under these conditions more often than is realised and it is being missed. Indeed the whole valve mechanism is capable of so many changes, from hour to hour, that it is quite possible a certain degree of emphysema may occur from time to time.

It appears reasonable to conclude that emphysema is not seen more often in company with positive pressure cavities owing to a conjunction of, and interplay between, the factors enumerated above, the whole being largely dominated by the difference in the rate of gaseous diffusion in the pulmonary alveoli as compared with a cavity. In

addition, the maximum valve effect is probably to be found in the region of the broncho-cavitary junction.

Referring to the second problem, whether or not the normal bronchial movements are abolished by tuberculous disease of the bronchi, there is little evidence one way or the other. Pinner (53) merely states "How much ulcerated or infiltrative lesions impair the inherent motion of the bronchi, and therewith their drainage, is not certain". As has already been said Ameuille & LeMesque allude to paralysis of the musculature of the bronchus draining a cavity. Such a bronchus, they state, remains immobile when viewed behind the X-ray screen: its lumen does not alter under any respiratory influences, including coughing. It must be extremely difficult, however, to make such an estimation by this means; consequently one is inclined to accept their view with considerable reserve.

In tuberculous infection of the bronchi Coryllos (29) refers to there being a loss of elasticity due to the more or less advanced tuberculous lesions present. This handicaps inspiratory enlargement and elongation of the affected bronchi, which in turn limits the inspiratory expansion of that portion of the lung.

An appreciable loss of the contractile and expansile movements of the larger bronchi in tracheo-

bronchial disease is not a feature which has come to be recognised by bronchoscopists. The movements of the larger bronchi are governed by the same principles as those of the smaller which, as already shown, consists of a passive dilatation on inspiration due to increased negative intrapleural pressure and a combined elastic recoil and muscular contractive movement on the part of the bronchial wall on expiration. Nevertheless it seems reasonable to suppose that a bronchiole which is markedly inflamed to the point of its walls being turgid, might assume a certain amount of rigidity. In addition, the bronchiole possesses a greater proportion of muscular tissue than does the larger bronchus. The efficiency and contractile powers of the muscle fibres would be expected to be interfered with at times by the inflammatory process with the result that the bronchiole would cease to undergo its normal movements.

Whilst preserving an open mind on this question perhaps the most satisfactory conclusion is that either state might prevail in a diseased small bronchus. The draining bronchus might continue to expand and contract with respiration and this movement might give rise to a check valve effect. In another case, the bronchioles might remain relatively impassive when no such valve effect would be created unless by some other agent such as secretions. Lastly, in view of the explanations already given it is perhaps no longer necessary to call upon such a theory to help to explain the absence of emphysema around a positive pressure cavity.

Narrowing of the bronchial lumen is caused by conditions other than swelling of the mucosa. The bronchial secretions themselves may cause obstruction. With destruction of the ciliated epithelium, the steady passage of mucus along the bronchi towards the hilum is interrupted. In addition the secretions become particularly thick and tenacious. Jackson (33) describes the endobronchial secretions, in contradistinction to sputum, as being of very high viscosity and adhesiveness, clogging the cilia and adhering to the bronchial walls. Jackson classifies these secretions amongst the endogenous foreign bodies capable of causing valvular obstruction to the bronchi. Samson (63) similarly describes the sputum in tuberculous tracheobronchitis as being exceptionally tenacious, mucoid, sticky and rubbery.

In addition to the obstruction caused by stagnation of secretions in a narrowed bronchial lumen the expulsive cough mechanism is reduced. Jackson has observed that prolonged contact of the bronchoscope with the bronchial mucosa produces tolerance of the latter to the cough reflex, presumably by fatigue of the afferent sensory nerve endings. Foreign bodies, he considers, may act in a similar manner.

As the endobronchial disease progresses, secretions, granulation tissue, sloughs and sequestra

of cartilage from the bronchus may be added to the obstruction already present. These, mixed with similar granulation tissue, sloughs and purulent secretions from the diseased lung or a cavity may form a very effective barrier in the form of tenacious secretions and solid plugs. As a result the bronchus may become blocked totally, or intermittently with the bronchial movements.

As healing sets in, further obstruction may occur from scar tissue contraction. This in itself might act as a permanent or intermittent form of obstruction. Even a relatively dry, fibrous stricture will be capable of acting as a ring valve, open on inspiration but tightly closed upon expiration and capable of producing a positive pressure cavity distally.

Maier (41) remarks upon the frequent occurrence of multiple tension cavities in the same patient and conjectures that this might be attributable to some systemic cause, such as a diffuse alteration in the bronchial wall or an element of bronchial spasm.

It cannot be stressed too often when considering the behaviour of a cavity, that the governing mechanisms are complicated and apt to undergo sudden

and frequent changes. Eloesser draws attention to the thinness of the dividing line between total and partial obstruction of a bronchus, and points out that slight complicating factors, such as a little more or a little less swelling of the mucosa, a little more or a little less viscid sputum, may tip the balance backwards or forwards between dilatation in the lung and atelectasis. The dividing line is equally thin between partial or intermittent obstruction, and full patency of the bronchial lumen. A cavity in which the internal gas pressures are equal to those of the atmosphere might suddenly be converted to a positive pressure cavity due to a slight increase in the swelling of the bronchial mucosa. A temporary accumulation of the bronchial secretions might convert the intermittent occlusion into a permanent one for a matter of hours, days or longer, when a positive pressure cavity will rapidly be converted to a negative pressure cavity owing to the absorption of its contained gases, with the result that the cavity will shrink in size or close for a shorter or longer period. Pinner summed up these conclusions when, alluding to this subject, recently he wrote (53) "The various events described must be understood as typical examples and it must be realised that bronchial occlusion and stenosis are not necessarily and not commonly static

phenomena. Many occlusions are temporary and intermittent; many stenoses are changeable in degree. Hence, the individual case frequently does not show the clear-cut phenomena, as described, but composite, changing and abortive pictures."

From what has been said it will be seen that the lumen of the bronchus draining a cavity may be in one of three states: I.Open; II.Closed; III.Inter-:mittently open and closed - meaning open on inspiration, closed on expiration. It will be realised that another form of intermittent opening and closure might occur by periods of alternation between I and II.

Most authorities are agreed upon what happens to a cavity when state III occurs - the cavity is inflated by a positive pressure. Around the question of the consequence of complete patency or closure of the draining bronchus there has been a great deal of controversy.

Before examining this question in detail it is necessary to realise that a cavity is the complex product of pathological and mechanical processes. The effects produced by the state of the draining bronchus form a part, and only a part, of this combination: a cavity cannot be regarded solely in the light of one or other of these processes.

Morland (45) describes two main factors responsible for cavity formation: destruction of tissue and inflation of air. It might be added that provided inflation does not always imply creating a positive intracavitary pressure (which strictly speaking it does), these two factors are common to the origin of all cavities. There is a great divergence, however, in the subsequent course of behaviour of cavities, a divergence which forms the basis of their classifications.

In consequence, it becomes necessary at this stage to adopt one of the many classifications of cavities in order to follow the behaviour of a cavity under the effect of different states of the lumen of the draining bronchus.

Coryllos classifies cavities simply according to the state of pressure of their contained gases. Whilst Pinner recognises that the state of the draining bronchus and the elastic stress of the lung play an important role in cavity behaviour, he classifies them simply according to three grades depending upon the amount of connective tissue formation in their walls. Because it is rather more comprehensive than some, the classification given by Ornstein and Ulmar (29) will be taken for the present purpose. These authorities allow more latitude than

Coryllos and classify cavities according to six categories. These are governed largely by the state of the draining bronchus but an independent place is also given to the pathology in and around the cavity wall.

Ornstein and Ulmar recognise: I. An early cavity formation with thick, ragged, irregular walls, the result of softening and sloughing of tissue and independent of the state of the communicating bronchus.

II. A single cavity, the chronic progressive cavity, which has a wall of granulation tissue in which the tuberculous enlarge and caseate, and the cavity slowly progresses in size. The bronchus is usually patent.

III. A third form which depends a great deal upon the condition of the draining bronchus. Because of granulation tissue formation and thick secretions within the bronchus a check valve is created which distends the cavity with air. There may be little original loss of tissue and little evidence of any peripheral invasion around the wall of the cavity.

IV. A cavity with a smooth wall and an open bronchus. The tuberculous process in the cavity wall has ceased, and become cicatricial.

V. A cavity with a thick wall and a closed bronchus. The walls are thick and avascular. In consequence no gaseous exchange takes place through

the cavity walls and the cavity remains unchanged. The cavity resists collapse therapy.

VI. A type misnamed a "bronchiectatic" cavity, frequently seen in retracted lung and giving an appearance of bronchiectasis on bronchography. At post-mortem the bronchi are seen to open abruptly into the cavities, which may be multiple, and which are in the lung parenchyma and are not bronchial dilatations.

It must be borne in mind, as Pinner emphasises, that lesions which affect the lumen of the bronchus interfere not only with aeration but with the drainage of the secretions of the bronchus and, it follows, of the cavity, as well.

I. THE EFFECT UPON A CAVITY OF AN OPEN BRONCHUS.

When the draining bronchus is open, the secretions from the cavity and bronchus are able to pass freely towards the upper respiratory tract to be expectorated. At the same time air is permitted to pass to and from the cavity, with the result that the internal pressure of the latter will be the same as that of the atmosphere.

Such a cavity will depend entirely for its closure or enlargement upon the extracavitary forces and pathological processes at work in and around it walls.

Depending upon the reaction of the tissues of the host to the tuberculous process, the cavity will belong to the second or the fourth of the categories described by Ornstein and Ulmar. If the disease is permitted to progress in the region of the cavity, further excavation will occur - the cavity conforming to type II. If, on the other hand, the tuberculous process has been checked and the cavity walls have largely healed, the fourth type of cavity will be found. In this type, the active manifestations of tuberculosis have disappeared, leaving in their wake a potential source of exacerbation of the disease. If, as Pinner and Parker describe (51) (53),

progressive, concentric scar shrinkage of a cavity walls can indeed take place, such a cavity will slowly shrink in size and ultimately may heal leaving a fibrous scar. On the other hand such a cavity might be prevented from shrinking by interfering mechanical factors such as Pottenger describes (see p.20) - powerful extracavitary forces of resistance. In time, the walls of such a cavity would probably become thickened with connective tissue and, even though the bronchus became closed at any stage the cavity might still be unable to close. Its walls would be incapable of yielding because of extracavitary forces and their own rigidity. In consequence the principles governing the absorption of gases would be unable to operate.

Thus it may be said that a cavity with an open bronchus and under conditions of immuno-biological stability would only be able to close by a process of scar tissue retraction in its walls, unless prevented from doing so by interfering mechanical factors. Where such factors were present the cavity would be held open by main force and would remain as a smouldering source of infection from the tubercle bacilli harboured in its ragged walls.

On the other hand it is generally accepted that such a cavity might heal biologically - that is by a

process of epithelialization of its lining wall from an ingrowth of healthy bronchial epithelium. Such a process has been described by Pagel and Simmonds (49), Auerbach and Green (4), Coryllos and Ornstein, and Pinner (53), amongst others.

As a result of open healing, there may be complete elimination from the cavity walls of all tuberculous elements capable of causing a recrudescence of the disease. A patent cavity would still appear radiologically but the patient would be sputum free or the sputum would be T.B. negative.

Such a state of affairs would, nevertheless, be unreliable. The likelihood of every trace of the tuberculous process being eliminated from the walls of such a cavity must be slender. Should evil days befall the patient a smouldering spark might light up the disease once more. Pinner maintains that such a process is rare and is liable to be incomplete: it is excessively rare not to find tubercle bacilli in sputum or gastric contents of patients with radiologically demonstrable cavities. Jaffé (29) pointing out the desirability of closing all cavities refers to the possibility of latent infection lurking in small tubercles or caseous areas in the healed cavity wall; even mentioning the possibility - which surely must be remote - of carcinoma arising from islands of meta-

:plastic epithelium.

The aerobic conditions which prevail in an open cavity are favourable to the growth of the tubercle bacilli within that cavity. The anaerobic conditions created when the bronchus is closed are unfavourable to these organisms.

Coryllos (18(19)(29) has analysed the gaseous contents of cavities under different conditions of bronchial occulsion. This has been done by needle aspiration. He found that in cavities with an open bronchus there was 16 - 19% oxygen, and 0.5 - 1% carbon dioxide (19 (29) (2% & 2.3% carbon dioxide (18)). In a cavity with a closed bronchus the oxygen content fell within a few hours below 1%, or disappeared; whilst the carbon dioxide content rose above 5%. The composition of atmospheric air is 20.96% oxygen, and 0.04% carbon dioxide (8). When the figures for the analysis of gaseous contents of cavities are compared with those of atmospheric air, it will be seen that the amount of oxygen in a cavity with an open bronchus approximates to the amount of oxygen in the atmosphere. In a cavity in which the bronchus is closed however, the oxygen is rapidly absorbed; whilst carbon dioxide diffuses into the cavity space from the venous blood raising the percentage of that gas within the cavity.

Coryllos & Ornstein (20), maintaining the bronchial origin of all cavity changes, hold that aeration is responsible for the thickness of the cavity walls upon which Pinner bases his classification. The thickness of the walls is due to tissue reaction which in its turn is dependent upon whether the tubercle bacilli are thriving in the oxygen or dying for want of this gas (see p.139).

With free drainage of the cavity, any risk from retention of secretions with secondary inflammation is removed. Further, there is nothing within the cavity to prevent apposition of its walls and healing with a minimal scar.

Thus a cavity with a patent draining bronchus will remain unclosed unless the pathology of its walls is such that they are capable of scar retraction and provided that this is not forbidden by the extracavitary forces. A thin-walled cavity with a patent bronchus would shrink with collapse therapy but would not close unless its walls had sufficient properties of scar retraction to fill the space due to destruction of tissue, always provided other means of cavity closure did not intervene. Conditions within an open cavity will be favourable to the growth of the tubercle bacilli which are probably

seldom absent. This will prolong the healing process and offer a constant threat of danger from bronchogenic spread of the disease.

II THE EFFECT UPON A CAVITY OF A CLOSED BRONCHUS.

By a closed bronchus is meant one which remains closed for an appreciable length of time as opposed to intermittent opening and closure. A bronchus might be closed for an hour or less, several days, or permanently. The effect produced will depend upon the length of time the closure persists.

The cause of closure may be extrabronchial or endobronchial.

Extrabronchial causes of closure.

Extrabronchial factors bring about occlusion of the bronchus by compression, by permitting the bronchus to collapse or by causing it to be kinked.

Bronchial compression in tuberculosis occurs mainly in association with enlarged hilar glands or when the bronchus is incorporated in the shrinking process of a region of massive fibrosis. In both of these instances the bronchial walls are compressed from without.

Therapeutic collapse of the lung more often acts by permitting the bronchus to collapse rather than by causing actual compression, except perhaps in the case of the marked degree of fibrosis which may follow a thoracoplasty. The mechanical basis of

collapse therapy is reduction of both lung volume and intrapulmonary tension. This is achieved either by reduction in the volume of the hemithorax, as for example by phrenic paralysis or thoracoplasty, or by direct reduction in the volume of the lung by means of pneumothorax. Reduction of lung volume and tension result in a corresponding reduction in the bronchial volume and tension. It follows that any condition which increases the intrapulmonary tension, such as consolidation - whether it be fibrotic, pneumonic or atelectatic - will produce an increase in bronchial volume, with bronchial dilatation. This will occur in the bronchi remote from the consolidation, situated in the region of the lung in which compensatory pulmonary expansion has taken place.

In collapse therapy, where there is a reduction in lung, and hence bronchial, volume and tension, contraction of the bronchial tissues is permitted owing to the elasticity of the bronchial walls. But in a normal bronchus this will never proceed beyond a reduction in the lumen: the bronchus will not close.

Collapse therapy can seldom be regarded as causing compression of the intrapulmonary tissues. Although lung volume and tension are reduced, the intrapleural pressure remains subatmospheric, or at

least at zero, until the lung is atelectatic and can shrink no more. This applies even in the case of a thoracoplasty. It is incorrect to regard the latter as a compression operation if by that is implied imposing a positive pressure on the intra-pulmonary tissues. Such a pressure is probably imposed only under such conditions as tension pneumothorax, massive pleural adhesions and, to a lesser degree and selectively, by a positive pressure artificial pneumothorax. A distinction must be drawn however between the immediate effects of a thoracoplasty and the late effects such as the compression by scar tissue retraction as a result of the operation.

In endobronchitis where the mucosa is already swollen, or the lumen partially occluded by granulations, a relatively small amount of relaxation in the extrabronchial tension might result in complete occlusion of the bronchial lumen. This would not have happened to a normal bronchus. A diseased bronchial wall furthermore, will be weakened by necrosis, which may include the cartilage plates.

The lumen might be closed by shrinking of the bronchus. This is sometimes associated with the primary complex but in this case compression of a

bronchus, rather than kinking, from the enlarged hilar glands is more likely to occur.

The distorsion might be produced by pulmonary consolidation, particularly fibrous consolidation and retraction. This is even more liable to occur in conjunction with collapse therapy. One section of a bronchus might be displaced in one direction by scar tissue retraction, and another section of the same bronchus might be displaced in the opposite direction by the regional displacement of the lung parenchyma as a result of collapse therapy. The bronchus falling between the two forces would become bent or, especially if diseased, kinked.

In this combination of factors the result will depend upon the locality of the retracting pathological process and the type of collapse therapy employed. The normal forces of movement of the lung are directed in the longitudinal plane of the bronchi, and tend to keep them straight. This is not altered by a uniform collapse towards the hilum as in the case of an uncomplicated artificial pneumothorax. But this is not the same as a distorted collapse due to pleural adhesions. Stivelman (68) believes that the position and direction of the bronchi, especially the smaller ones, are affected by pneumothorax collapse and that

such a procedure by causing a change in the position of the lung might kink the bronchi. This authority apparently believes that this might take place even with an uncomplicated pneumothorax collapse. As in the case of a distorted pneumothorax collapse, kinking of a bronchus might be produced by elevation of the base of the lung following phrenic paralysis or dropping the apex with a Semb thoracoplasty.

Holcombe & Weber (32) consider that it takes a fairly strong force to kink the bronchial tree other than the bronchioles, and add that it is the larger bronchi that lead to the larger cavities. They consider that a thoracoplasty or a hydropneumothorax might be forceful enough to produce this effect but not an artificial pneumothorax - not even a positive pressure artificial pneumothorax. Aufses (5) believes that kinking of a normal bronchus, as for example by lobar displacement following collapse therapy,might cause partial obstruction, or such a procedure might convert a previous partial obstruction into an intermittently complete obstruction. Complete obstruction of a bronchus as a permanent condition is rare, according to this authority. With a thoracoplasty obstruction might occur in a bronchus already partially obstructed by granulation tissue or fibrosis. Aufses reports two cases in both of which artificial pneumothorax resulted in marked

increase in size of a cavity. Bronchoscopy revealed narrowing of the bronchial lumen possibly due to some extrabronchial factor in one case, and kinking of the bronchus in the other case.

It is likely that the result depends upon the combination of the factors at the time - such as the type and amount of disease of the bronchus and the nature of the distorsion - rather than upon the strength of the distorting forces.

Endobronchial Causes of Closure.

Endobronchial occlusion might be caused by swelling of the bronchial mucosa, granulation tissue, an accumulation of secretions, or by necrotic debris - including cartilaginous sequestra. The secretions and debris may be held up and, becoming more or less inspissated, form a plug which may become tightly wedged within the airway. Auerbach and Green (4) maintain that the lumen at the broncho-cavitary junction is usually narrowed, in which case it is easy to believe that proliferation of granulation tissue in the bronchus might proceed until the opposing surfaces have joined. Where closure does not occur the authors consider it is probable that continued infection of the granulation layer keeps pace with, or ahead of, proliferation of the granulation tissue; or continued tuberculous infection of the surfaces of the occluding lumen will not permit

closure. Further closure of the broncho-cavitary junction may be favoured by contraction of the fibrous ring at this area. Bronchial or peribronchial fibrosis might gradually further occlude the bronchus in the manner just described, and as described by Holcombe & Weber (32), until final and complete pathological healing and sealing of the lumen is accomplished. Coryllos & Ornstein (20) believe that fibrous peribronchitis due to connective tissue replacement of muscle tissue is the most important factor in the permanent, definite closure of bronchi and in the healing of cavities. The end result of such a process will be either a localised fibrous occlusion, or conversion of varying lengths of the bronchus into a fibrous string as illustrated by Loesch (40) (figure II). Healing by fibrosis in this manner is the ideal to be hoped for in all cases of cavity closure - apart from early healing with apposition of the cavity walls - and is the one form which ensures, as far as possible, permanent closure of the cavity.

The work of Loesch is of particular interest. Not only does he demonstrate complete and permanent healing obliteration of the draining bronchus, but further light is thrown on the mechanism of cavity closure. The work is a pathological study of complete

closure of cavities in four cases treated by artificial pneumothorax; one other treated by bed rest; and a sixth, transitional case, demonstrating marked cavity shrinkage with bed rest. In four of the cases death had been due to disease in the other lung; one was a pneumonectomy specimen. Loesch considered that careful serial sections are indispens-:able for accurate results: examination limited to a few sections can be misleading. This was shown in the study in a case where a bronchus which appeared to terminate near the capsule of the focus was discovered to by-pass the area and curve round it. The results from this work can best be understood by reference to figure II. The main conclusions Loesch draws from his study of the histological sections is that when the lung is freed from the pull of the chest wall by collapse therapy - as with an artificial pneumothorax - retraction and finally collapse of the lung occurs due to its elasticity: any cavity present is included in the process, provided no bronchial obstruction occurs to give a tension cavity. The mechanism leading to cavity closure in these cases was for the most part one of shrinkage of the cavity with simultaneous contraction, approximation and finally fibrous obliteration (1) of the cavity outlet, or (2) of the tuberculous bronchus at the point of transition into the normal portion of the

bronchus, or (3) at both of these points when caseous matter is retained in the diseased portion of the bronchus between them. In each instance, caseous matter was trapped in the contracting cavity and became inspissated, completely filling the lumen.

The effects of closure.

Once the bronchus has been completely closed, air or secretions contained within the bronchus or cavity distal to the block, are trapped. What follows is a subject for considerable disagreement amongst authorities. Coryllos and Bloesser, who hold opposite views as to whether or not this causes cavity closure, both claim to have the support of the majority of authorities.

In 1932 Coryllos presented his conception of the mechanics and biology of cavities. This may be stated simply as follows: after the evacuation of caseous matter from a tuberculous lung, a hole is left which communicates with a bronchus. The cavity thus formed is maintained patent because of the difference between the intracavitary and extracavitary pressures, the former being atmospheric, the latter subatmospheric on account of the negative intrapleural pressure. If the bronchus leading to the cavity is occluded, the air trapped in the cavity will be absorbed by the principle of obstructive atelectasis, and the cavity will close. If the lumen of the

bronchus is narrowed, then, on account of the inspiratory dilatation and expiratory contraction of the bronchus, a one-way valve will be created which will result in distension of the cavity and air spaces placed distally, by a principle of obstructive emphysema. Thus far, it will be noticed, the views of Coryllos summarise the greater part of the principles which have been put forth herein. Coryllos however, does not exempt the alveoli from emphysema when a cavity is distended in this way.

But Coryllos soon after goes on to state that if this conception is correct, the most important factor in cavity closure is the bronchus and not the cavity wall (see page 39). By 1939 he even goes so far as to maintain that the complete closure of the bronchus is the only mechanism by which tuberculous cavities may heal, whatever may be the size and nature of their walls, and even when anatomically they cannot collapse (20).

The main principles set forth by Coryllos are founded upon sound laws of physics and biology; and his views have been corroborated by analysis of the gaseous and bacterial contents of cavities and cavernoscopic investigation of the living subject. It would appear that unquestionably the majority of authorities agree largely with Coryllos, though by no

means with the whole of his beliefs (Ornstein & Ulmar (29)(32)(68)(71)(4)(53)(16).

It is necessary, however, to examine the evidence produced by some of the authorities who disagree with Coryllos on the subject of the effect produced upon a cavity by complete closure of the draining bronchus.

Salkin, Cadden & McIndow (61) sought to show that "cavities may exist in the lung without any communication with the tracheobronchial tree, and in this state, may enlarge, absorb air or fluid and be productive of physical signs." Within an hour after death the authors placed the cadaver in an upright position before an X-ray screen, then proceeded to introduce into the bronchial tree by way of the trachea, and occasionally directly through the chest wall into the cavity in addition, a radio opaque substance less viscous than lipiodol. By manual compression of the chest wall they sought to promote the distribution of the opaque substance throughout the respiratory tract and into the alveoli. As a result of subsequent X-ray appearances they assess whether or not the draining bronchus communicates with the the cavity. There were 70 cases treated in this manner, for this particular purpose, and in 37 of these cases

post-mortem was afterwards performed. Out of a total of 147 cavities over 3 cms. in diameter, in these 70 cases, it was found that 21 (14%) did not communicate with a bronchus. Of these apparently blocked cavities 15 were examined at post-mortem and as a result the authors described four causes of blockage: (1) intracavitary block, (11) extra-bronchial fibrosis, (111) bronchial plug and (IV) inflammatory block due to tuberculous bronchitis,

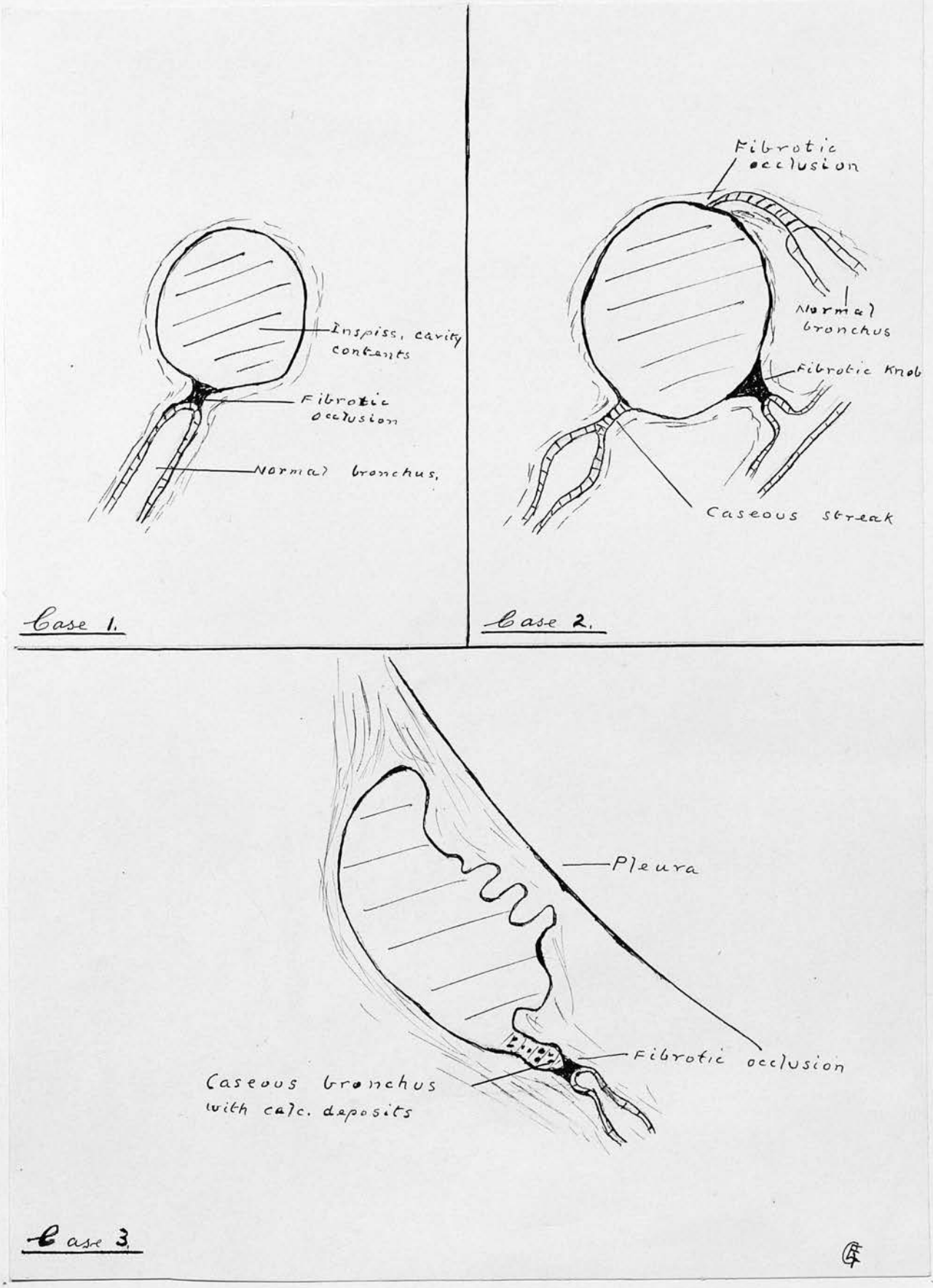
(I) Intracavitary block was described in 3 cases. Here the cavities were tightly filled with fibrino-gelatinous debris so that the medium could not enter.

(11) Extrabronchial fibrosis was described in 2 cases and consisted of dense fibrosis and cicatrization which incorporated the bronchi and the cavity wall.

(III) Obstructing bronchial plugs were found in 3 cases, and consisted of a tenacious mixture of pus, mucus and debris firmly fixed in the lumen of the bronchus near the cavity opening. These could not be dislodged by rough manipulation during the injection of the dye.

(IV) Tuberculous bronchitis was found to be the commonest cause: this was noted in 7 cases, in which there was found to be mucosal inflammation and swelling which occluded the lumen. At the border

"Closure of Tuberculous Cavities."

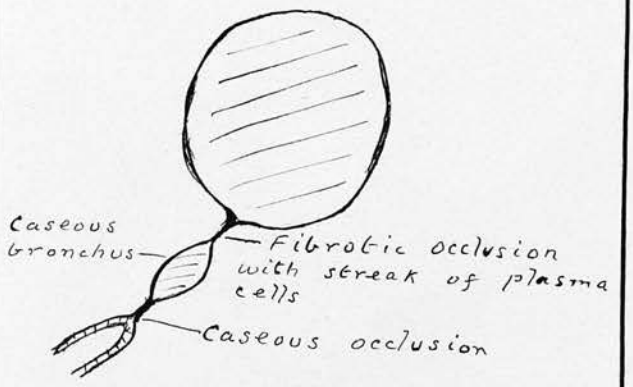


Diagrams of pathological cases described by Loesch, Am. Rev. Tubere., 1944.

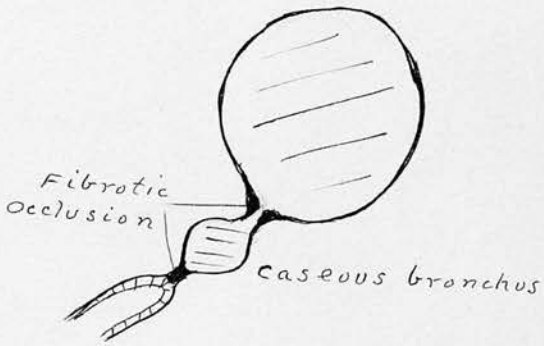
Figure II

(Copied from Loesch (40))

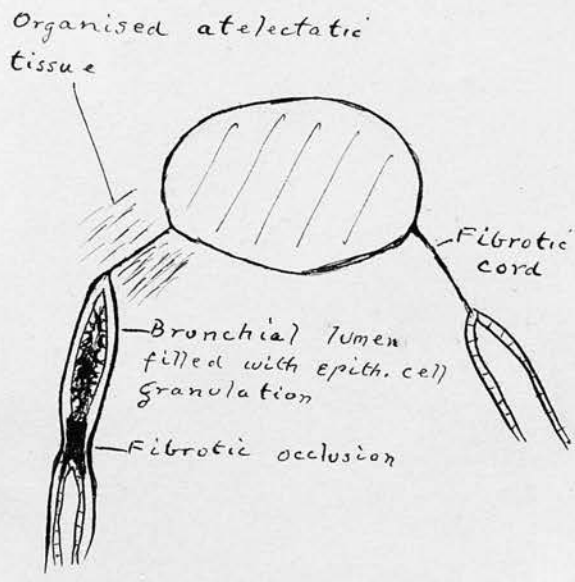
"Closure of Tuberculous Cavities."



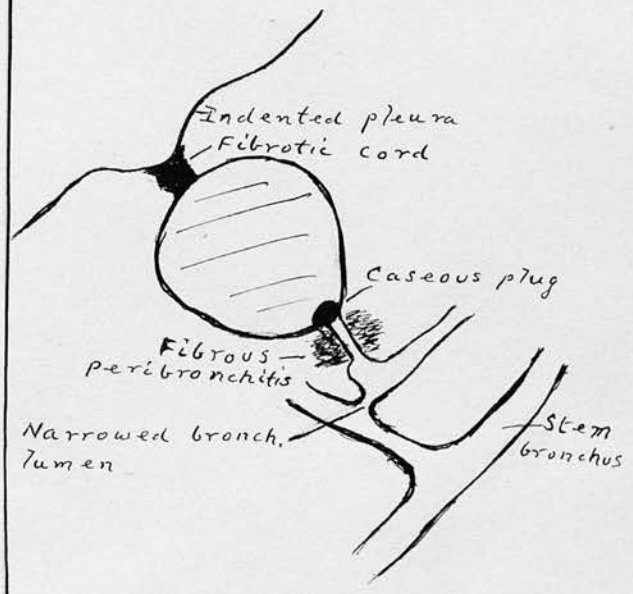
Case 4. Formerly a definite cavity.



Case 4. Formerly a caseous focus with commencing cavitation.



Case 5.



Case 6.

Figure II

Loesch (40) (contd)

Auerbach & Green (4).

The impression throughout, including the clinical studies quoted by Salkin, is that a check value mechanism was in operation in most of these cases. Of the so-called blocked cavities, serial X-rays prior to death had shown that 10 cavities had increased in size - some rapidly , 9 had been stationary for months, one had decreased in size, and one had shown rapid variations in size. Of the 10 cavities which had increased in size ante-mortem, 5 were subsequently examined at post-mortem: of these 5, there were 4 found with signs of inflammation of the bronchial mucosa and one with a plug. A plug was also found post-mortem in a case where ante-mortem the cavity had shown rapid variations in size. It would appear very suggestive that in life a check valve mechanism, rather than total bronchial occlusion, had been present in a large proportion, if not all, of the 10 cavities which had increased in size ante-mortem; and in the one which had shown rapid fluctuation in size. As will be shown later, the conditions described by Salkin, in II, III and IV are very liable to produce, in life, intermittent bronchial occlusion, with the effect of an inflationary check value.

A further objection is that although the authors claimed that the opaque medium they used was

capable of going anywhere that the air could go, it is expecting too much of such a fluid substance to believe it was capable of finding its way reliably through the sticky secretions within the lumen of a stenosed bronchus and so into a cavity. This is especially so when the difficulty is imagined which some pathologists (4) (71) must have experienced hunting with a probe until the track leading into a cavity was finally discovered. A similar problem is presented when an attempt is made to induce fluid to enter an ampoule with a long, narrow neck. The air trapped in the ampoule blocks the way. This was realised by Salkin and his co-workers but they believed that the method they employed was largely able to overcome the difficulty. The authors quote Sante, who pointed out that when an oily dye is employed for a bronchogram the alveolar air is trapped and, at first, only the bronchi are filled. The circulating blood, however, absorbs the air in the alveoli and the oil is drawn into them. This, they admit, cannot happen in the case of a cadaver: but their success in this direction they consider must indicate that alveolar filling is governed by the viscoscity of the medium employed and the amount retained - not coughed up. Sante's explanation would appear to be nearer the mark, however.

The authors admitted (62) that dissection afterwards showed that a large number of the alveoli had not filled with dye, even when filling appeared dense on X-ray. This they ascribed to air being trapped by dye in the manner just described. Even with improved technique - less viscous solution, slower injection and manual pressure on the chest to force the air out - they were only able to effect "an almost complete alveolar filling in many cases, as seen at autopsy".

As in the case of the alveoli, the most successful way of inducing the dye to traverse the narrow passage and enter a cavity is by intracavitary suction - employing a negative pressure within the cavity, in other words. A strong negative intrapulmonary pressure cannot be produced in a cadaver by the means employed in these investigations. Furthermore, expulsion of air from these "blocked" cavities by manual compression of the chest wall, as a prelude to creating a negative intracavitary pressure, was most likely prevented by the check valves. In short, no reliance can be placed upon successfully working the dye into a cavity by rough manipulation of the chest wall as carried out by these authors.

It might be argued that it is unnecessary to demand a standard of complete alveolar filling in order to decide whether or not a bronchus leading into

a cavity is blocked. This objection might be countered by a further unwitting confession on the part of the authors. Im 12 cases diagnosed by this method as block-:age of major bronchi, and in numerous blocks of smaller bronchi many of which latter were of long standing, there was no accompanying atelectasis. Furthermore, in 63 cases of bronchiectasis (? employing a slightly different dye) there was no alveolar filling in 61 cases. "Certainly", they conclude, "the absence of alveolar filling indicates that little or no air reaches these alveoli, but, instead of showing atelectasis, they appear normal, or show emphysema or pneumonitis. This fact further strengthens the theory of interlobular air channels. Such an area had not only a stagnant respiratory circulation, but probably has also a stagnant vascular and lymphatic circulation." Alternatively this might further serve to demonstrate the uselessness of bronchography as a means of tracing out details of the endobronchial pathway.

From the report that these authors give, there is no suggestion that bronchography was employed chiefly as a preliminary means for sorting out those cavities which did not definitely possess a patent draining bronchus. Post-mortem search is not indicated as having been the chief means of detecting whether or not the bronchus was patent: and it is perhaps significant that subsequent post-mortem examinations do not appear to have contradicted

bronchography findings in a single case. How careful a search was made it would perhaps be wrong to judge. There is however no indication that a meticulous search was made with a probe for an opening into the cavity after the manner of Vineberg and Kunstler or Auerbach and Green. The suspicion arises that had this been done, an opening would have been found.

Finally the authors stress the absence of alveolar filling dependent upon bronchiectatic bronchi, and refer to the occlusion produced by pus. They do not differentiate between temporary blockage by agents such as pus and the permanent occlusion associated with fibrosis or, less permanently, caseous plugs.

Thus it is concluded that the experimental evidence produced by Salkin, Cadden and McIndoe in an endeavour to disprove the theory that closure of the draining bronchus results in closure of the cavity is founded upon unreliable experimental methods. Being such, their methods cannot be aceepted as a basis upon which to found such important conclusions. In consequence their views are, in the main, worthless.

Vineberg and Kunstler (71) similarly doubt the efficiency of the technique adopted by Salkin, Cadden and McIndoe. In their experience, the bronchial tract leading to a cavity could always be found provided sufficient care was given to the search. In three of their cases, where the pathologist had previously failed to find the com-

municating tract, a bronchus was eventually found which admitted a probe quite easily into the cavity. In the process of entering, the probe lifted away from the cavity floor a trapdoor-like flap of tissue beneath which was revealed the mouth of the draining bronchus. In two of these cases a thoracoplasty had failed to close a positive pressure cavity.

Brunn et al (16) examined the bronchi in approximately 100 autopsies on tuberculous lungs. In no case did they find a closed bronchus leading to a cavity.

Auerbach and Green (4) also disagree with Salkin and his co-workers in that they used cadaver experiments and did not distinguish between permanent and transient block. These authorities have observed over two thousand cavities and as a result state "...we have never seen an open cavity with a diameter of 3 cm. or more in which we were unable to trace, with a slender probe, a bronchus opening into the lumen of a cavity."

Kayne, Pagel and O'Shaughnessy (34) appear to have been impressed with the work carried out by Salkin. The only objection they raise is that the time interval between cavity formation and blockage of the bronchus, or between blockage of the bronchus and bronchography, was not taken into account by Salkin and that this "militates against his conclusions".

Eloesser, whilst strongly advocating the check valve hypothesis of cavity inflation, condemns complete bronchial occlusion as a means of cavity closure. This authority (23) accepts the view that in total bronchial obstruction there is absorption of alveolar air with their consequent collapse but elsewhere (24) expresses the view that total permanent obstruction would lead to a cavity resisting attempts at closure. He produces no evidence in an attempt to confute Coryllos' hypothesis that a blocked cavity will close by a process of obstructive atelectasis, and the experiments he carried out on persistent cavities (24), when needling them and injecting lipiodol and methylene blue, produced nothing at all convincing to support his objection. Eloesser holds that when the draining bronchus is blocked there is often a retention of secretions within the cavity: as these seldom remain aseptic, this leads to fever, toxicity and disaster. Eloesser (24) mentions the experiments of Salkin but makes little comment. The inference is, however, that he looks upon their findings as substantiating his own views.

Writing in 1931, Pinner (51) made no mention of the state of the draining bronchus having anything to do with cavity behaviour. Fibrosis in, or attached to the cavity wall, played a prominent part in this respect and, depending upon the nature of its

relationship to the cavity wall, was capable of exerting an expanding or a contracting influence upon the cavity. "The interplay of caseation, liquefaction, resorption and fibrosis," Pinner wrote, "with the peculiar mechanical conditions existing within the pulmonary parenchyma account for the striking structural differences of cavities at a given time in their development." Rapid changes in size of a cavity - enlargement and closure - were to be noted frequently but the only attempt Pinner made at an explanation was to assume that the elastic tension of the lung exaggerated the size of the hole made by loss of tissue and promoted its spherical shape. This explanation, however, does not cover fluctuations in size of the "accordion" variety of cavities which were noted by this authority.

Saley (60) writing at about the same time and expressing considerable agreement with the views of Pinner, similarly made no mention of the state of the draining bronchus playing any part in cavity behaviour.

Originally Pinner denied the probability that bronchial occlusion might result in cavity closure. By 1940, however, he partly admitted that such a denial was no longer tenable in the light of more recent knowledge(52). He now admitted that bronchial occlusion could close a cavity but he still

associated with the process, though not to such a marked degree as formerly, complications as a result of secondary infection within the cavity. A large percentage of cavities, he believed, contained secondarily infected organisms which when bottled up within the cavity might produce serious complications. Furthermore Pinner was very mistrustful of "blocked" cavities which he considered were very liable to open up again. When this occurred, the bronchus being stenosed, the cavity became even more difficult to close again. A cavity should only be considered healed when anatomically it was incapable of re-opening. A cavity might heal with or without closure of the draining bronchus but he considered that to maintain that bronchial occlusion was the only method of cavity closure - as did Coryllos - was open to doubt. Pinner believed that a fibrosing process was often at work with slowly contracting scar tissue playing a prominent role. The two pathological cases he described in support of this latter view are most convincing and will be referred to in more detail later.

Thus by 1940 Pinner appeared to have arrived at a more balanced conception of the mechanism of cavity closure than was the case with Coryllos whose approach to the subject remained predominantly one-sided.

When Pinner published his views in 1945, shortly before his death, it is probably true to say that the opinions he expressed with reference to the effect of bronchial occlusion upon subsequent cavity behaviour coincide in the main with those which are universally accepted at the present time. There is no very great departure from the views he expressed in 1940. The role of the bronchus assumes perhaps a slightly more prominent place: whilst the incidence of secondary infection is perhaps no longer regarded as being as high as was formerly maintained - nevertheless its dangers are in no way minimised. The bronchus has by now established for itself a secure place in Pinner's mind as being one of the major factors influencing cavity behaviour. Bronchial occlusion is still represented as being followed by cavity closure or the complications associated with secondary infection but there is now a third possibility described - the cavity may become filled with caseous matter and, if the bronchial occlusion persists, shrink, with the inspissation of its contents. Cavities heal as formerly described by occlusion of their draining bronchi and subsequent absorption of air or by a process of slow scar tissue contraction initiated by the fibrous tissue in the cavity wall.

The effect of the retention of secretions within a blocked cavity, and especially in connection

with secondary infection, has aroused considerable controversy. Secretions with a pure tuberculous infection in a cavity with a closed bronchus behave like the contents of a cold abscess. When secondary organisms are present however an active state of affairs often prevails with toxicity, fever, localised inflammation and suppuration. As has already been shown, Pinner and Eloesser believe that secondary infection is a real danger as a complication of blocked cavities. In the opinion of the former (53) the collapse of a cavity which has an occluded bronchus is in many instances interfered with by the pathological action of these organisms.

That occlusion of the draining bronchus does at times lead to troublesome complications is generally accepted. Whether or not the cavity is secondarily infected on these occasions must frequently remain unknown. Lilienthal (39) describes a case in which the bronchus draining a tuberculous cavity became angulated or plugged as a result of distorsion following a paravertebral thoracoplasty. The patient at once became very toxic with high fever. Rib resection and blunt entry of the now putrid abscess were resorted to in order to give free draining to the abscess contents, whereupon the symptoms subsided and the patient made a satisfactory recovery.

Fischel (28) believes that with actively secreting cavities, premature obstruction of the draining bronchus by means of collapse therapy, leads to retention and enlargement of the cavity (see page (190-191).

Jackson (33) advocates bronchoscopic examination of cavity secretions prior to instituting collapse measures in order to determine the nature of the organisms present. Tubercle bacilli require aerobic conditions in order to survive; pyogenic organisms are indifferent; whereas Jackson very much doubts the wisdom of collapsing a lung, thereby possibly occluding a draining bronchus, if anaerobic organisms are going to be shut in. Reference has previously been made to the fact that gas analysis of closed cavities shows that conditions within such a cavity are unfavourable to the tubercle bacillus (page 74).

Auerbach and Green (4), and Coryllos and Ornstein do not believe that bronchial occlusion is liable to result in complications from secondary invading organisms. Coryllos and Ornstein (20) maintain that the idea of the prevalence of this complication has been based upon post-mortem investigations. According to these authorities Rassfeld found anaerobic organisms upon culture of the heart blood in 100% of cadavers where there were

ulceronecrotic intestinal lesions: Faragó found anaerobic organisms, including B. Welchii, in 100% of post-mortem cavities a few hours after death, but in only 12% of cases in sputum of living patients. As the autopsy cases had had advanced disease, and as such cases generally have intestinal affection, Faragó's cases were of a type comparable with those of Rassfeld. Coryllos and Ornstein conclude from these findings that cavities are contaminated post-mortem from the bacillaemia and therefore do not portray a true picture of the state of affairs to be found in life. Coryllos investigated this problem in seven living cases. Needle aspiration of cavities in these cases was performed by Petroff, Director of the Laboratory, who employed a scrupulously careful aseptic technique. As a result of these investigations, pure cultures of tubercle bacilli only were obtained: there were no secondary organisms. These findings were corroborated under similar conditions by Bedall at the Veterans' Hospital, Castle Point, New York. These results and the absence from the literature of references to toxic symptoms resulting from the accumulation of secretions in blocked cavities, led the authors to conclude that a cavity can more often be likened to a pure tuberculous empyema rather than to a mixed infection tuberculous empyema.

It is pointed out, nevertheless, that Coryllos only investigated seven cases: and Faragó did find anaerobes in the sputum of 12% of living cases.

In connection with this problem, Pinner (53) declares that he has seen many non-acid fast organisms in smears made post-mortem from cavity contents but they can rarely be found in the cavity walls.

Thus it would appear that the incidence of secondary infection of cavities is still a matter for a good deal of speculation. Pinner (1945) sums up by saying "there are no reliable data available to indicate what percentage or what kind of tuberculous cavities are secondarily invaded". (53). Nevertheless, like Eloesser, Pinner believes that this condition is prevalent enough to warrant that it should be regarded as a real source of danger: Auerbach and Coryllos, on the other hand, disagree with such a view.

Whatever the truth may be regarding secondary infection of cavities, for practical purposes there is no evidence so far which demands an unduly cautious attitude being adopted towards seeking bronchial occlusion as a means of cavity closure.

Whether complications arise only as a result of secondary organisms or whether they may arise with a pure tuberculous infection, it would appear that a

reasonable practical means towards avoiding complications would be to give the patient a period of bed rest before commencing collapse therapy: this would apply especially if the cavity is large and there is much sputum. Such a means will give every opportunity for the tuberculous process, and any other infective process, in the region of the cavity to become less active: this would apply whether the secondary organisms were aerobes or anaerobes and whether the disease was limited to the cavity walls or included the surrounding parenchyma. With the disease less active there would be less liability for the cavity to be so productive and in need of a free drainage path for the secretions: at the same time exacerbation of the disease would be less likely to occur.

Advanced stages of occlusion, up to complete closure of the draining bronchus, are frequently associated with the retention of caseous matter within a cavity and its broncho-cavitary junction. This, with progressive inspissation and calcification of the caseous matter, is recognised by Pinner (53), Auerbach and Green (4), Loesch (40), Pagel and Simmonds (49), and other authorities as one of the common manners in which cavities heal. Pinner (53) states that of 39 cavities, found healed at autopsy, which Pagel reports from his own observation and the

literature, 22 were healing by this method. Their ultimate fate was unknown: some would have gone on to real healing by inspissation and sterilization of the caseous mass, Pinner believes, whilst others would have re-opened and discharged their contents through the unblocked bronchus. Bronchial occlusion in these cases was reported as being either by caseous bronchitis, which might lead to fibrous occlusion, or by a caseous plug: thus the occlusions, as Pinner points out, were by reversible or irreversible processes. Paget and Simmonds, as a result of post-mortem studies, concluded that previous bronchial occlusion sometimes, at least, played a leading part in this mode of cavity healing.

A bronchus may be closed for a long or a short time. It may finally become sealed permanently after the manner demonstrated by Loesch (fig.II) in which case the cavity can be regarded as being truly closed. On the other hand closure of the bronchus, and with it the cavity, may be but temporary. It is this latter feature, which is so common with cavities, that has given rise to a general attitude of mistrust towards all "closed" cavities. Temporary closure might be produced by a bronchial plug as a result of which air in the cavity will be absorbed. After the course of hours, days, weeks or longer the plug might be dislodged thereby opening the lumen once again.

the same effect might be produced by the subsiding of an inflammatory or allergic swelling of the bronchial mucosa or the straightening out of a bronchial kink. Before full patency of the bronchus is secured, however, intermittent occlusion with the bronchial movements is very liable to occur in many instances, causing inflation of the cavity with a positive pressure.

This curious behaviour of cavities in which they shrink and expand alternately, often rapidly, has been described by numerous authorities. Mantoux refers to such cavities as "accordion cavities". Andrus (3) observed radiologically one thousand cavities, measuring them from time to time, and was surprised to note that it was rare for a cavity to be the same size in two consecutive films. As a result of recording the measurements of some of the cavities studied radiologically at the end of this thesis it is pointed out that allowance has to be made for the discrepancy between the real and the radiologically apparent size of cavities.

To regard any cavity as being permanently closed must always be a qualified assumption. Provided there is a spark of infection left smouldering in a cavity scar or in the immediate vicinity of the healed cavity focus, an exacerbation of disease might

result in renewed cavitation. It might not be the same cavity which re-appears but the results are much the same.

A cavity which has healed by apposition of its walls leaving a scar must be regarded as being the ideal type of healed cavity. Auerbach and Green (4) believe that this form of healing is not common. It would be expected to occur, however, in cases of early healing of a thin-walled cavity. These authors believe that cavity healing with inspissation of caseous contents is the most frequent method. Pinner (53) regards such "blocked cavities" as being virtually cold abscesses and considers that they frequently re-open, sometimes years after having become blocked: eventually they may really heal. Brunn et al (16) believe that such inspissated cavity contents may be coughed up leaving a cavity again. It is not difficult to imagine how the contents of any one of the cavities illustrated in figure II, and which have recently been described as "truly closed" cavities (excepting case 6), might rupture into another bronchus or one or other of the former draining bronchi following exacerbation of the disease in that focus.

In conclusion, permanent closure of a cavity must always be regarded as an uncertainty. It is evident that closure of the draining bronchus is an important factor in causing cavity closure by absorption of air, with sealing off and subsequent inspissation of the cavity contents: no valid evidence has been produced to contradict this belief. Such a process is infrequently accompanied by immediate complications, which are probably due to secondary infection of retained secretions. Later complications are from dissemination of tuberculous caseous contents following exacerbation of the disease. The day-to-day fluctuations in size of a cavity are most likely due to changes in the intracavitary static pressures brought about by changes in the degree of bronchial occlusion. The bronchus may be now patent; now stenosed causing an inspiratory check valve of inflation; or again completely closed with resulting closure or shrinkage of the cavity following absorption of its contained air, only to be re-inflated when the obstructing secretions have been removed by the combined efforts of cilia, bronchial movements and the blast of air and tussive squeeze associated with coughing.

III. THE EFFECT UPON A CAVITY OF AN INTERMITTENTLY CLOSED BRONCHUS.

By intermittent bronchial closure is meant an intermittent closure of the bronchial lumen with each expiration. This is invariably of such a nature as to promote a one-way check valve to the passage of air. Some valve mechanisms permit air to pass distally only and so produce inflation; other valves allow the air to pass out of the lung only and so produce deflation. The mechanism is dependant upon three factors related to the bronchus: (1) the respiratory movements of dilatation on inspiration and contraction on expiration, (2) the air currents and changes in pressure within the bronchus during respiration and (3) the pathology as it affects the bronchial lumen.

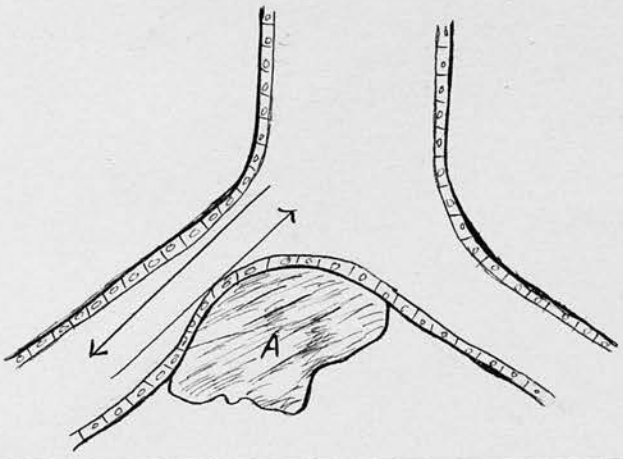
In view of what has already been described previously the first two factors need no supplementing. All that need be said of the third factor is that it embraces all the different pathological conditions, both endobronchial and extrabronchial, which have already been described with reference to the causes of complete bronchial closure. In intermittent closure their development has not reached a stage at which complete occlusion of the bronchus is attained through all phases of the respiratory cycle.

Chevalier Jackson (33) claimed to be the first to have observed valvular bronchial obstruction. This was over thirty years ago with reference to foreign bodies found in the bronchus on bronchoscopy. Soon afterwards he demonstrated that "endogenous foreign bodies in the form of pathological tissues and exudates commonly do the same thing." These "endogenous foreign bodies" may be secretions of high viscosity, sloughs, sequestra of cartilage or enlarged glands. A swollen bronchial mucosa will further contribute to the process by narrowing the lumen. Jackson soon realised an important point which Salkin overlooked. Prior to the introduction of bronchoscopy, post-mortem examinations had shown tissue changes, or products of such, which were capable of causing occlusion of the bronchus with resulting atelectasis. "But," said Jackson, "looking into the living moving bronchi revealed the fact that bronchial obstruction in the living is valvular and that the autoptic type of occlusion is only one of four types of obstruction" - adding that valvular obstruction of bronchi is dependant upon physiological movements that cease at death and is consequently not in evidence at post-mortem. By way of challenging any contradiction Jackson pointed out that he was not offering a theory but reporting on what is in evidence objectively at any bronchoscopy clinic.

Having given a description of tuberculous disease within a bronchus as seen through the bronchoscope, and finally having described the pinkish granuloma overlying the tuberculous ulcer and more or less filling the lumen, Jackson (33) then goes on to explain that any remaining lumen might be seen in one of four conditions: (I) it may remain open on inspiration and expiration; (II) it may open enough to admit air on inspiration but close immediately at the beginning of expiration; (III) a small mass of exudate, pus, caseous material or debris may be seen to move up on expiration and slap down on the beginning of inspiration, both movements being due to the current of air; (IV) the entire lumen may be occluded throughout the whole respiratory cycle. These appearances, said Jackson, are endoscopic evidences of valvular mechanisms causing emphysema or atelectasis.

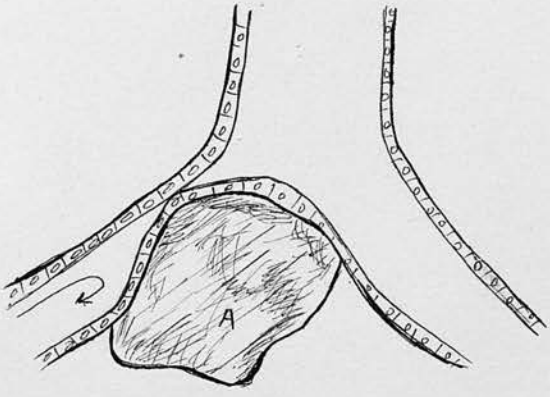
Jackson recognised four types of valve mechanism: a stop valve, a by-pass valve, a one-way valve of inflation, and a one-way valve of deflation. The condition just described under (I) is an example of a by-pass valve according to Jackson's differentiation, whilst (IV) is an example of a stop valve. But it is pointed out that neither in a by-pass valve nor in a stop valve is a true valve mechanism at work. In one

(1) BY-PASS VALVE

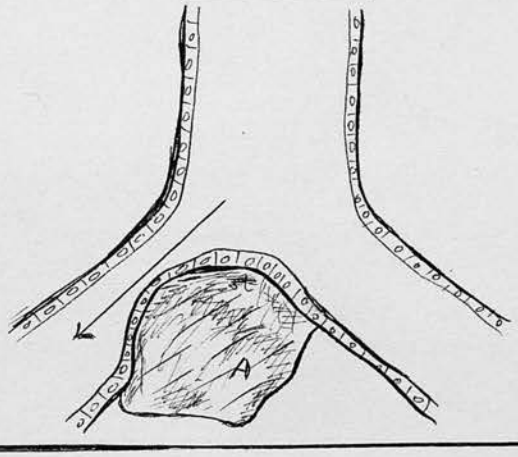


(2) CHECK VALVE

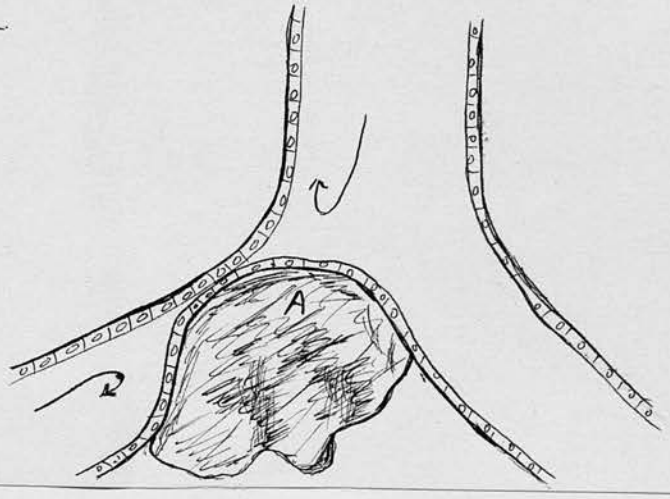
EXPIRATION



INSPIRATION



(3) STOP VALVE



Peribronchial lesion — tuberculous gland (A) — causing various degrees of progressive obstruction.

(These are some of the valve mechanisms described by Jackson. Only one is a true valve (2))

Figure III

(copied from Jackson (33))

there is a completely open though narrowed bronchus and in the other a closed bronchus - in both cases throughout the whole respiratory cycle. It will be realised, however, from Jackson's description that with increasing swelling of the mucosa, growth of granulation tissue or enlargement of a hilar gland, a bronchial stricture will become a bronchial check valve and eventually a total occlusion (see fig. III)

The different types of true bronchial or broncho-cavitary valve mechanisms which might occur will best be understood by reference to the illustrations in figures IV & V. They may be described as one-way valves created by the following:

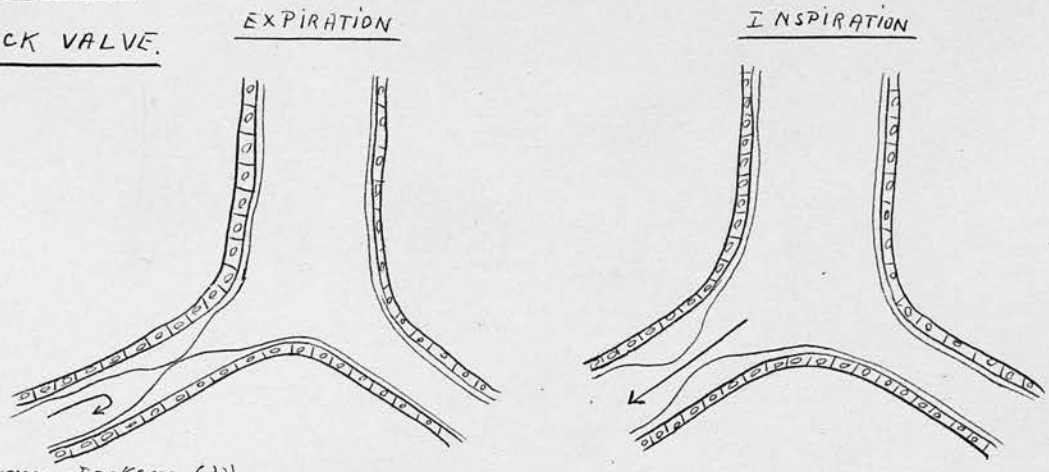
(1) The respiratory movements of contraction and dilatation of a bronchus the lumen of which has been narrowed by some pathological process.

(2) The stump of a bronchus projecting into a cavity.

(3) A plug which moves to and fro with respiration, impinging upon a stenosed ring of the lumen of the bronchus in the process.

(4) A bronchus entering a cavity wall obliquely in such a manner as to create a flap valve.

(a) EXPANSILE CHECK VALVE.



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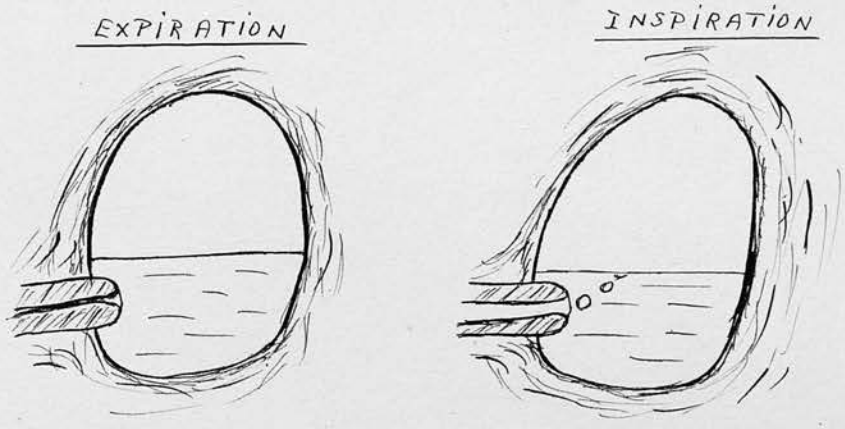
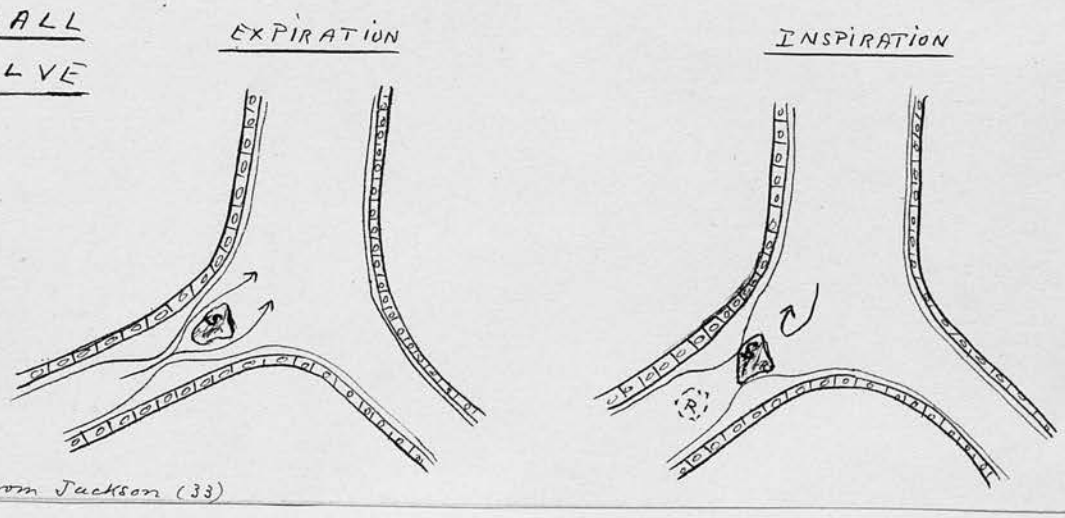
(b) BRONCHIAL STUMP VALVE



(c) BALL VALVE



Copied from Jackson (33)

Fig. IV True Bronchial Valves.

(a) The valve closes on expiration owing to contraction of the bronchial lumen and swelling of the mucosa. The result is inflation of the parts distally.

(b) The bronchial mechanism is similar to (a). Secretions in the cavity also obstruct the outlet on expiration.

(c) Small mass of secretion (s) acting as a valve with the mucosal oedema, causes obstructive atelectasis. The plug is swept back into the socket on inspiration.

(1) The Expansile check valve(figs. III(2) and IV(a)), as Jackson calls it, has already been described. Its mechanism depends upon stenosis of the bronchial lumen in conjunction with the respiratory movements of the bronchial walls. The conditions which cause the stenosis have already been described and at this stage it is only necessary to repeat that these causes may be endo-, peri- or extrabronchial. The commonest forms are mucosal swelling, granulation tissue and fibrous stricture. Enlarged hilar glands may cause similar effects though with more likelihood of emphysema. In addition, kinking or distorsion of a bronchus might result in the lumen being diminished and increased in size with the respiratory movements.

According to Jackson, the expansile check valve is the commonest type of check valve encountered. It has been recognised by most authorities.

(2) The Bronchial Stump valve.(fig.IV(b)) Owing to the resistent nature of its walls a bronchus will often withstand the necrosing process which is causing the cavity. Charr, Woodrow and Burgess (17) studied cavities in 131 patients, 39 at autopsy. They described some of the cavities as being criss-crossed by cord-like structures which in many cases were

bronchi and their branches. Jaffé(29) has described a similar appearance given by the larger blood vessels - thrombosed veins and arteries with periarteritis. When such bronchi eventually slough away a stump may sometimes be left projecting into the cavity. This, Charr believes, may act as a valve by the expiratory contraction of its narrowed lumen. Furthermore, the stoma of such a bronchus may dip below the level of the retained secretions. When this happens air will bubble through the secretions to inflate the cavity on inspiration but on expiration the stickey secretions will fill the opening which is below the level of the fluid and prevent the escape of the air. Morland(45) described a case in which there was a large cavity with a fluid level but no sputum. After bed rest and phrenic paralysis the cavity disappeared leaving a small scar. The author considered it difficult to explain the fluid level with absent sputum if the bronchial outlet was patent. The small scar on healing was suggestive of inflation rather than destruction of tissue. Morland believed that secretions were forming a valve in this case in the manner just described. Coryllos and Ornstein(20) as a result of the pathological examination of giant cavities, described some of the bronchi entering a cavity on a small papillary projection or, the reverse, at the bottom of a small funnel.

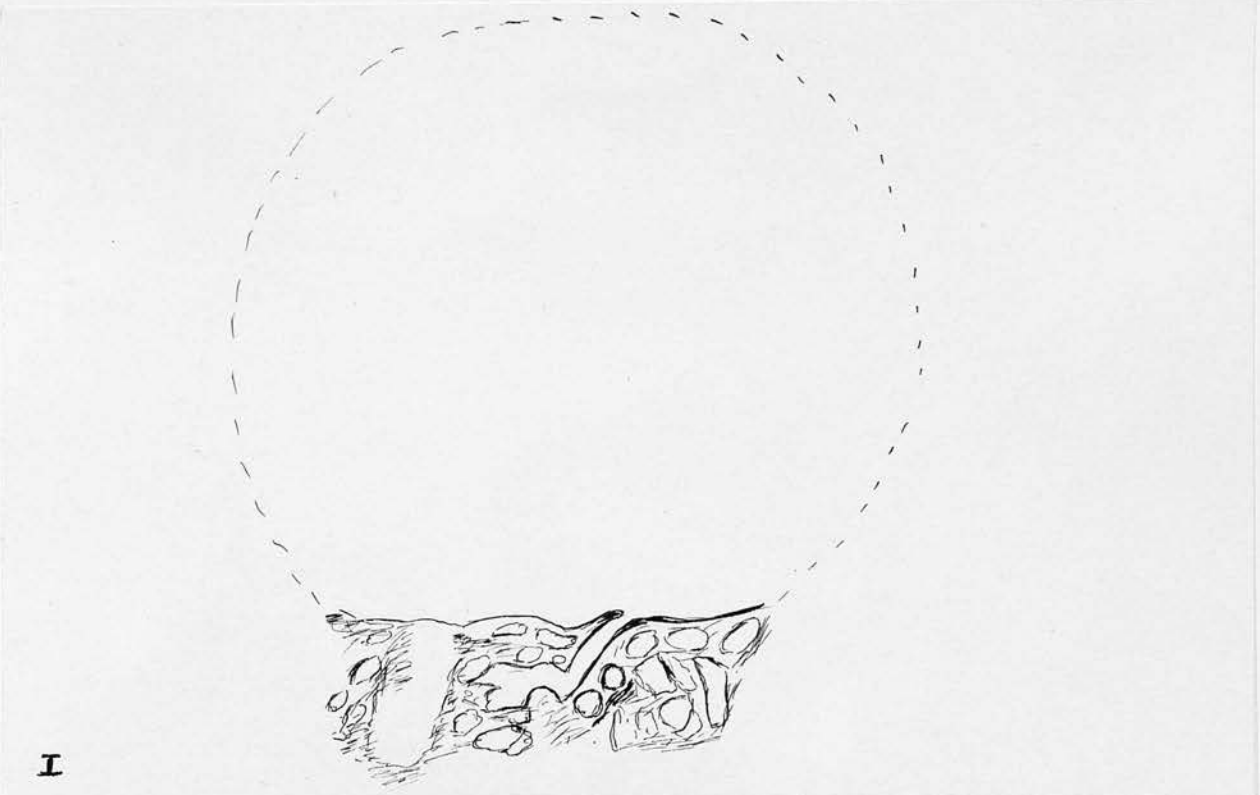
(3) The Ball valve(fig.IV(c)) has been well described by Jackson. The factors responsible for creating this type of valve are stenosis of the lumen and a plug of necrotic tissue or caseous matter which is capable of being pressed against the orifice of the stenosed portion of the bronchus by the current of air and held there by the relative positive pressure created behind it. When the reverse process of respiration commences the pressures either side of the plug change with the result that the obstruction is swept back to its original position and the valve is once more open. The plug may be a small collection of secretions of high viscosity or of inspissated caseous matter, a small blood clot, slough or sequestrum of cartilage, or a small loose flap of tissue attached near the valve area.

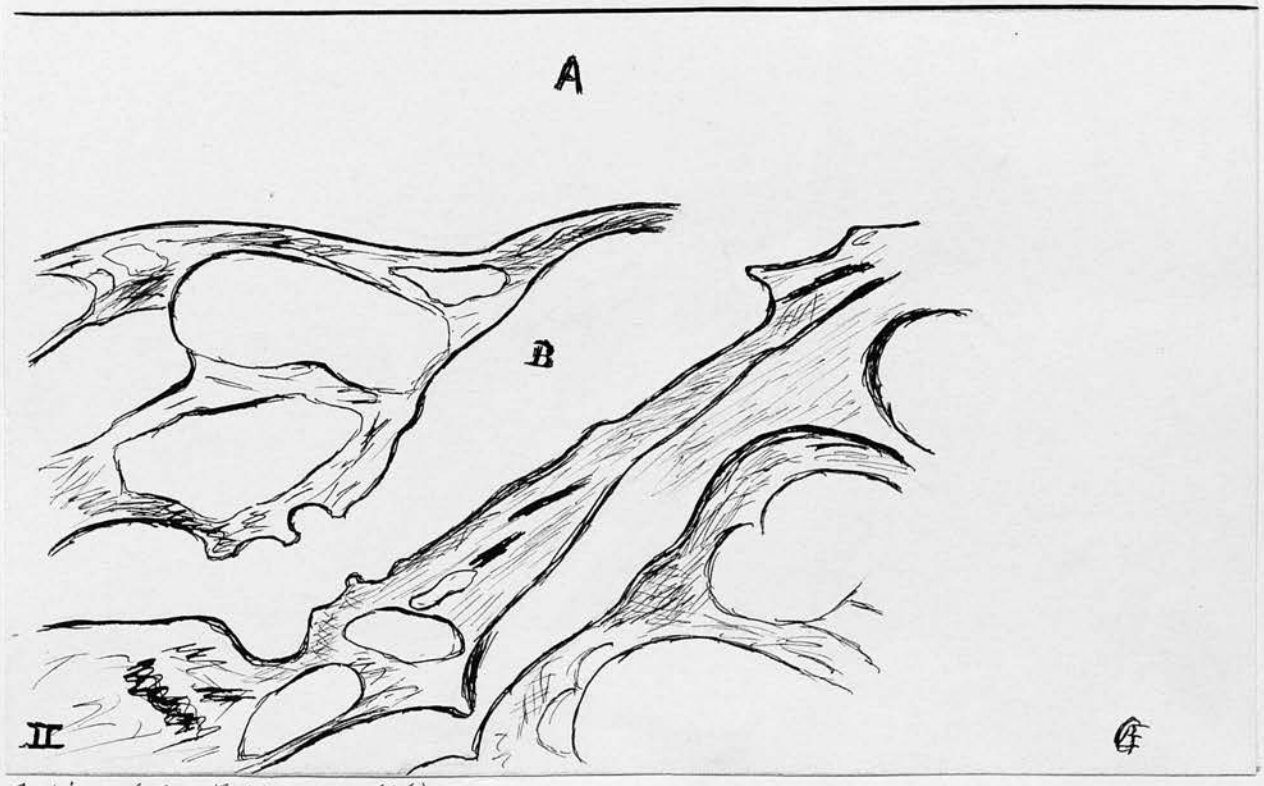
As shown in figure IV(c) the valve will be an inflator or a deflator depending upon which side of the stenosis the plug is to be found.

(4) The Oblique Bronchus valve(fig. V) has been described by Coryllos, Ornstein(20), Eloesser(25), Kjaergaard(36) and others. In this type of valve the bronchus traverses the cavity wall at an oblique angle thus creating a flap valve out of the overlying

portion of the floor of the cavity. Coryllos likened this type of valve to the mouth-piece of a saxophone. Eloesser described such a valve which was discovered at autopsy: the bronchus ran for some distance through the cavity wall entering the cavity on a slant, being shut, according to the author, when the intracavitary pressure rose. This case is of interest from a pathological standpoint, but the fact that experimentally the cavity was a tension cavity - pressure rose to +7 with the bellows andfell slowly to +2 - is of little value owing to the experiments having been performed on a cadaver(see page 42).

The classical description of this type of valve mechanism has been given by Kjaergaard. This authority studied spontaneous pneumothorax in the apparently healthy and came to the conclusion that the condition was caused by the rupture of a large subpleural vesicle. Kjaergaard studied pathological sections of the bases of three such vesicles. In two of the cases he was able to demonstrate, with excellent illustrations, the valve and explain its mechanism. In the first case a bronchiole was found approaching the base of the vesicle at an angle of 45^{o}. The valve was created by a tongue of emphysematous tissue(see fig.V). In the second case a small bronchus approached the vesicle making an





Copied from Kjaergaard (36)

Fig. V True Bronchial Valve. (Oblique bronchus valve)

I X 5. Base of subpleural vesicle. A little to the right of the middle, a respiratory bronchiole is seen running obliquely into the floor of the vesicle. An emphysematous tongue flap above the bronchiole acts as a valve being pressed down to occlude the bronchiole when the pressure in the vesicle rises.

II X 30 Same vesicle as I. A = lumen of vesicle. B = bronchiole.

These diagrams are of the emphysematous tongue flap valve demonstrated by Kjaergaard (36). II is a copy of a microphotograph. The second case described by Kjaergaard is similar but with a fibrous tissue tongue flap.

angle of 30° with the base and there was a tongue of fibrous tissue making a flap valve. In each case the overhead flap was part of the floor of the vesicle. In the third case it was not clear by what mechanism the valve effect was created. Kjaergaard concluded that such valve effects are caused by localised tissue distorsion owing to scar retraction or to localised emphysematous changes without scarring. In both instances flap valves may be created in the manner described. A tremendous pressure can be built up within such a vesicle which eventually might burst resulting in a spontaneous pneumothorax. Kjaergaard refers to the earlier similar findings of Hayashi and Fischer who studied three post-mortem cases. Hayashi found microscopically a valve-like structure resembling the flap valve of a pump.

It is easy to see how an increase of pressure within a cavity governed by such a valve would cause the tongue-flap to be pressed down, thus closing the outlet to the bronchus.

Consideration of the different valve mechanisms just described will reveal that there are two main mechanical principles underlying the function of all these valves. Broadly speaking bronchial and broncho-cavitary valves may be classified as (I) those which

are governed by the movements of contraction and dilatation of the bronchial wall - which may be termed bronchial movement valves - and (II) those which are governed by the pressure of air upon the valve - such may be called pressure valves. In this latter group, the greater the air pressure the more efficient will be the valve.

In the first group, the bronchial movement valves, the valve opens every time the bronchus dilates on inspiration. A strong positive pressure within a cavity governed by such a valve, as after coughing, will be released the first time the bronchus dilates - this will be with the next inspiration. Such a cavity will be unable to empty itself each expiration and there will always be a residue of trapped air under a slight mean positive pressure. Any undue positive pressure will quickly be relieved. This mechanism probably accounts for the frequent slight but constant positive pressure which must prevail within many cavities helping to keep them patent and spherical in shape, but without undue distension (see p. 41). At present, however, such is an assumption - it is unproven.

In the second group of valves, the pressure valves, the state of affairs is different. Once a

sufficiently strong positive pressure has been created within the cavity the valve is shut and no movements of the bronchus will induce it to open. The ball valve(3) and the oblique valve(4) which have recently been described, are in this group. These are the valves which are probably often responsible for the true tension cavities.

It will be realised that a bronchial movement valve can approximate in its effect to a pressure valve, and vice versa. If the bronchial mucosa is very swollen so that it will only just admit air on full dilatation, as for instance during a paroxysm of coughing, such a valve will not open again during quiet respirations. Unless the air trapped in the cavity is under sufficiently high pressure to force itself through the occluded lumen the air will remain trapped at a fairly high positive pressure. On the other hand, an accumulation of secretions which are acting as a ball valve may, on occasion, be unable to hold in check a strong positive pressure, when the effect produced will be only a slight positive pressure within the cavity.

It will thus be seen that all stages of positive pressure can be promoted within a cavity by different

pathological conditions and by different degrees of these processes.

In view of the fact that secretions and necrotic matter so frequently accompany mucosal swelling it must be concluded that valve mechanisms of both groups must often be present together. Should the theory be correct that there is frequently a loss of the physiological movements of the bronchi in the pressure of tuberculous bronchitis then, in such cases, the plugs and secretions within the bronchial tract must be playing a prominent part if there is a positive pressure cavity present - apart from the slight predominance of the positive over the negative pressures which prevails in a cavity as a result of narrowness of the bronchial channel, and expiration being longer than inspiration.

A positive pressure cavity depends for its inflation upon the prior development of a negative intracavitary pressure. A small amount of air will be trapped during quiet respiration. The intracavitary pressure will fall just below that of the atmosphere, perhaps to - 1m.m.Hg on full inspiration, and a small amount of air will be drawn into the cavity. On expiration the pressure will rise perhaps to +1 m.m.Hg and air will commence to pass out of the cavity. If a bronchial movement valve is

present the bronchial walls will come together and so close the airway before the air has finished leaving the cavity with the result that a small amount of air will be trapped at a pressure slightly above that of the atmosphere. The amount may be very small and just enough to keep the cavity patent and spherical. If a pressure valve is present the air current flowing through the bronchus will push the ball valve into its socket, which is the orifice of the stenosed position of the bronchus. A small positive pressure will be built up behind the valve by the trapped air and this will maintain the plug or flap in its place until the balance of pressures changes with inspiration. If the valve mechanism is an oblique bronchus the raised intracavitary pressure may be just enough to cause pressure on the valve and so close it. In all these instances, during the next respiratory cycle air will only enter the cavity, or the bronchus distal to the valve, when the outside - or proximal - pressure is greater than the pressure of the trapped air. The peak period for this tendency for air to be admitted into the cavity will be at the height of the inspiratory phase when the trapped air will have reached its lowest pressure. The lower the pressure, the more the air that will enter the cavity.

It stands to reason that the amount of inflation under conditions of quiet respiration can not be very great. In order to produce a cavity containing gases at a high positive pressure conditions must be created in which a greater negative pressure is produced within the cavity in order to fill it with a larger amount of air. Forced respiration or coughing create such conditions because forced inspiration is included in such processes: this means the production of a high negative intrapulmonary pressure.

Coryllos and Ornstein(20) describe three phases in the act of coughing: (i) inspiration, (ii) compression and (iii) expulsion. A tension cavity will be unable to expand during the inspiratory phase if the positive pressure within is already higher than or equal to the intrapulmonary pressure created by the expansion of the chest. During the second phase, the compression of intrapulmonary air against a closed glottis prior to the actual cough, there will be no change in the amount of air contained in the cavity because the pressure is raised still further and the valve in consequence will remain closed. In the third phase, the explosive cough, the intrapulmonary pressure suddenly falls but the air will still be trapped in the cavity because of the valve. The cavity may

expand on account of the fall in the surrounding intrapulmonary pressure but no air will enter. It is the long drawn inspiration prior to a cough which by creating an increased negative pressure within a cavity causes the latter to inflate. If there is a bronchial movement valve this will open with the next inspiration and any undue pressure behind the valve will be released. In the true tension cavity, on the other hand, the pressure valve will remain shut during the ensuing respirations. As the air in the tension cavity is absorbed the pressure gradually falls until ultimately the cavity will become capable of admitting more air on inspiration.

This explanation is supported by the experimental evidence of Vineberg and Kunstler(71) who introduced needles through the chest wall into 54 cavities with a view to detecting tension cavities. They quote the following standard as a guide towards assessing tension cavities by the behaviour of the intracavitary pressures:

Initial pressure	-2 +2
End of inspiratory phase	-2, goes to 0.
End of expiratory phase	+2, remains steady.
Forced respiration	-4 +4
Quiet respiration	0 +4

Cough	+30
Quiet respiration after cough.	+6 +10
Withdrawal of air.	-2 +2
Introduction of air.	+6 +10

According to this standard the pressure in such a cavity during quiet respiration is approximately zero. Actions such as forced respirations and especially cough raise the intracavitary pressure. It must be concluded that it is the multiplicity of incidents such as the occasional deep breath or cough which by subjecting the cavity to small or greater amounts of inflation from time to time and for varying periods, eventually comprises the tension cavity. The result will be the balance between the inflation instalments on the one hand and the rate of air absorption within the cavity on the other.

It may be argued that coughing produces a high positive intrapulmonary pressure which will inflate a cavity by a backfire mechanism. To a slight extent this may be so. The reason why this is not more so is that during the compressive phase when the intrapulmonary pressure is raised against a closed glottis, the pressure both inside and outside the cavity rises uniformly. Air might be forced into a

rigid-walled cavity by such a means. The walls of a resilient cavity will respond to the extracavitary compression. In so doing, in accordance with Boyle's Law the pressure of the contained gases will be raised to withstand the increased pressure of air within the bronchus. If the walls are rigid, however, and show little response to the extracavitary compression there will be little change in the intracavitary gas pressure from this means with the result that air can be blown in from the bronchus. It might reasonably be concluded that the various degrees of thickness, and therefore rigidity, of cavity walls in general might cause a slight time lag and resistance to the extracavitary compression being communicated through to the intracavitary gases: the result would be that a small quantity of air might sometimes be forced into a cavity during the compression stage of a cough, before the intracavitary pressure had risen sufficiently to oppose the pressure of the air in the bronchus.

If the foregoing be true, straining against a closed glottis, as for example when lifting a weight or straining at stool or on micturition, will promote inflation of a tension cavity mainly by virtue of the increased inspiratory act associated with such efforts.

The experiments of Brunn and coworkers(16) tend to confirm the foregoing conclusions. These authorities made continuous Kymographic records of cavities during tanspleural decompression therapy. As a result of these findings they concluded that "changes in the intracavitary pressures are only infrequently related to the actual breathing of the cavity": and add that the amount of air which actually enters a tension cavity is very small; it does not enter bit by bit with the respirations but at infrequent intervals. By this method they were also able to demonstrate that air leaked into a cavity when a negative pressure was induced, and that the valve was 100% efficient when air was introduced into the cavity experimentally causing the pressures to rise to +5 +20, in which state they would remain for 5-30 minutes.

The efficiency of the pressure valves governing cavities was also demonstrated by Eloesser(24) and Coryllos and Ornstein(20). These authorities were able to show that the valve remained closed upon the creation of a strong positive pressure within the cavity and opened when suction was applied. Eloesser conducted his investigations on living subjects by

means of a needle introduced through the chest wall into the cavity. Coryllos was able to watch the proceedings through a cavernoscope. He injected saline into the cavity, which was retained even when the pressure was increased by the injection of air. It was sometimes difficult or impossible to see the orifice of the draining bronchus but upon applying suction air was seen to bubble through the saline from the hidden bronchial orifice. At the same time the valve had apparently been released for the saline disappeared, having flowed into the bronchus. As Coryllos pointed out, Eloesser was effecting the same valve release during some of his attempts to close cavities by transthoracic suction: he was simply drawing air through the cavity from the bronchial tree and failing to create any very great negative intracavitary pressure. At the same time, Coryllos pointed out, Eloesser was demonstrating that the draining bronchi of these cavities were not permanently and completely closed.

That a pressure valve is efficient and capable of causing a high positive pressure to be built up was shown by Kjaergaard(36) and by Alexander and coworkers(1). The latter describe a case in which a large ballooned cavity was situated in the right

upper lobe. The patient died suddenly following a two rib thoracoplasty. On opening the cavity at post-mortem there was a gush of air. No bronchial communication was found but the authors believed that a one-way valve must have existed which had been sufficient to cause cardiovascular failure from the pressure in the distended cavity acting upon the heart and great vessels. They considered that preoperative aspiration of air from the cavity might have avoided this calamity.

A tension cavity is frequently a fairly large cavity The size however, will be governed by the elasticity of the cavity walls and of the surrounding parenchyma, and by the magnitude of the positive pressure within the cavity. A cavity, the walls of which are considerably thickened by connective tissue, will be unable to dilate much even though the internal pressure may be great. A fairly recently developed cavity with little of the nature of a wall, and situated in a resilient and relatively healthy lobe of the lung, will be capable of a great deal of expansion. Thus the size of the cavity is no immediate indication of its internal gaseous pressure. Most authorities who describe "pressure cavities" or

tension cavities or the "cavernes ballonées" of the French writers have in mind the second type of cavity, with relatively thin walls. This type is sometimes met bulging into the pneumothorax space following collapse therapy, when it is an immediate warning of danger.

Bobrowitz(10) describes a case where right artificial pneumothorax had failed to close a small cavity after ten months with 80% collapse and apparently no adhesions present. The cavity had not been visible during this time but sputum had been persistently T.B. positive. No endobronchial disease was seen on bronchoscopy. The sputum became T.B. negative one month after partially letting up the pneumothorax and remained T.B. negative with the shallower collapse. The author believed that this was an instance of a small invisible tension cavity hidden in dense lung which had been allowed to close on re-expansion of the lung. Bobrowitz believes that the amount of air trapped and the degree of positive pressure in a cavity may be great or small.

This opinion of Bobrowitz is in accordance with the conclusions already reached. There is no dividing line between the different degrees of

positive pressure contained within cavities: there are only different gradations of pressures. Cavities can be described as being governed by one or other of the two classes of valve mechanisms - the bronchial movement or the pressure valve - and in the majority of cases there is a combination of the two.

It might be accepted as a reasonable hypothesis that the low positive pressure cavities are more likely to be governed by bronchial movement valves or valves created by secretions which are capable of only weak resistance to entrapped air pressures, whilst the high positive pressure cavities or true tension cavities are more likely to be the result of pressure valves or advanced degrees of bronchial stenosis.

The differentiation of the two groups of valves has little practical significance. Endobronchial disease features largely in both groups. Treatment will be directed mainly along the broader lines indicated by radiological and bronchoscopic manifestations in conjunction with clinical signs and symptoms.

Andrus(3) admits the soundness of Coryllos' hypothesis that cavities are closed by bronchial occlusion but considers that this is not complete and

final. He believes the idea that a valve mechanism is responsible for inflation of cavities is "entirely possible" but does not consider such a mechanism is easily or satisfactorily explained. Furthermore he objects that it is unnecessary to call upon such a device to explain the behaviour of cavities. Both the fluctuations in size and the closure of cavities can be explained, according to Andrus, on the basis of "atelectatic shrinkage of the regional diseased lung during the process of resolution and healing." Any hole contained within the atelectatic mass will be shrunk with it. A cavity is opened by being remotely placed in relation to the atelectasis which will thus pull it open. The explanation given for a positive pressure cavity is that the bronchus of a cavity which is being reduced in size owing to the atelectatic shrinkage of the pericavitary tissue, may be occluded: as a result the trapped air would be compressed. Andrus believes that "pericavitary atelectatic shrinkage is probably of greater frequency and importance in the closure of cavities than is the bronchial obstruction of Coryllos." Both factors, however, are considered to be frequently in operation, doubtless often in conjunction. "In fluctuations of regional atelectasis", Andrus concludes, "we have a sufficient explanation of the fluctuations in size of cavities as here recorded."

As has already been shown, Andrus does not recognise the significance and importance of the gaseous exchange which inevitably takes place within the tissues and the air spaces of the body, and consistently denies this physical process its rightful place. The intracavitary gases play a passive rather than an active rôle according to his conception: changes in the intracavitary gas pressures are the effect, rather than the cause, of fluctuations in the size of a cavity. The elastic pull of the lung and shrinking atelectatic tissue play the leading rôles in cavity behaviour, regardless of the state of the intracavitary gases and the fact that atelectasis is entirely the result of the absorption of alveolar air following closure of the bronchus.

Andrus admits, rather half-heartedly, the possible existence of valves; that cavities may be closed by occlusion of the bronchus; and that regional atelectasis may result from bronchial or bronchiolar obstruction. But fluctuations in bronchial obstruction would appear to be a more reasonable explanation of cavity behaviour than the fluctuations of regional atelectasis: regional atelectasis always follows a prior bronchial or bronchiolar block and is produced in

no other way. If the alveoli close in atelectasis, the same principle might surely be applied to a cavity.

It is concluded, therefore, that Andrus produces no valid argument against the belief that valve mechanisms such as have been discussed in this section, exist and influence cavities and cavity behaviour to a very marked degree.

In the opinion of Rafferty (55) none of the theories which have been put forward in an attempt to explain the mechanics of tension cavities is entirely satisfactory. If the bronchial factor is not the only factor, in Rafferty's opinion it is probable that at any rate it is frequently the most important.

THE CAVITY WALLS.

A cavity is a hole in a lung, the result of the elimination by way of the bronchial tree of liquefied necrotic tissue. The walls of a cavity consist of the surrounding lung parenchyma, which to a lesser or greater extent has undergone morbid histological changes as a result of the localised tissue response to the tubercle bacillus. In addition, as Monaldi has pointed out, a ring of atelectasis very frequently forms around a cavity or, it may be added, interposes itself amongst the pathological processes which constitute the cavity wall.

Pinner (51) (53) has classified cavities according to the nature of their walls. This classification has been widely accepted by authorities including Saley (60) and Keers and Rigden (35). Most of the more recent authorities, including Pinner himself, acknowledge in addition the influence of the draining bronchus in cavity behaviour. The classification according to Pinner (53) consists of three categories as follows:-

(1) Cavities without an established wall.

These are simply "holes in an area of caseous pneumonia." They show no evidence of possessing any granulation tissue or fibrous tissue which can be regarded as forming a cavity wall. Radiologically they are moth-eaten areas in regions of dense consolidation.

(II) Cavities with a young fibrous wall.

In these cavities the connective tissue fibres are not hyalinized and are developed as the encapsulation of a pneumonic area or during excavation. These are the cavities the regular outline of which formerly gave rise to the term "annular shadows". They consist of a central lining of necrotic tissue surrounded by a thin ring of circular connective fibrous tissue fibres and an outer zone of lung parenchyma or of tuberculous granulation tissue or exudative lesion. Pinner also describes strands of fibrous tissue which radiate like the spokes of a wheel from the ring of circular fibres. These radiating fibres follow the preformed connective tissue which surrounds the bronchi and blood vessels or which is found in the septal spaces. This type of cavity is particularly affected by the state of patency of the draining bronchus, which affects its size, shape and developement.

(III) Cavities with old, thick fibrotic walls.

In this group are found cavities lined by necrotic or granulomatous tissue surrounded by a thick layer

of fibrous tissue which is frequently hyalinized. These cavities are sometimes a later developement of the previous groups of cavities. They may, on the other hand, be late excavations of caseated foci which have been encapsulated for a long time before finally becoming liquefied and excavating.

The hole in the lung, or cavity, and the histological changes in the surrounding parenchyma which constitute the walls of the cavity are the outcome of three recognised tissue responses to the tubercle bacillus. As Pinner points out (53) the processes are fundamentally the same as any other tissue response to an invading organism where inflammation results. The reactions take the form of necrosis, exudation and proliferation. According to Rich (57), these processes are governed by the degree of resistance of the host to the invading tubercle bacilli, the number and virulence of the invading bacilli, and the degree of hypersensitivity within the host. As a result of liquefaction of the products of tissue necrosis and exudation, some of the diseased parenchyma will be eliminated by way of the bronchus, leaving a cavity. Should these destructive processes persist and predominate the

cavity will become progressively larger at the expense of the surrounding lung tissue. Proliferation, on the other hand is concerned with the formation of connective tissue in the cavity walls. This is a reparative process leading to encapsulation of the disease focus. As a result of the combination of three processes an encapsulated caseous focus often develops which may ultimately calcify, or, becoming liquefied and being eliminated through the bronchus, will leave a cavity.

From the point of view of the present study attention will chiefly be concerned with the process of proliferation and with the combined product - encapsulation of a caseous focus. A cavity with a blocked bronchus and containing inspissated caseous contents may also be included within the latter - the combined product. Although exudative processes and atelectasis take part in the formation of cavity walls these processes will not give to the cavity walls any properties of self-retraction which might result in cavity closure. If such properties are to be discovered in the walls of cavities they will be the products of the proliferative reaction.

From the point of view of cavity closure it is important to know whether or not the proliferative

changes are capable of producing a connective tissue which, by virtue of its own inherent proporties, is capable of bestowing upon the cavity walls the power of self-retraction. Monaldi (43) describes some cavities as having a true retractive power of their own. This is the result of two factors: a negative intracavitary tension due to closure of the bronchus, and secondly the elastic recoil of the cavity walls and pericavitary tissues which have been put on the stretch by the distension of the cavity.
Histologically, elastic fibres are found in the walls of some cavities. But elastic fibres can only recoil; they cannot retract less than their own length.
A distended cavity may recoil, if permitted, until the distension ceases, but the space left by the loss of tissue will not be filled in. As many authorities point out, frequently the actual loss of tissue may not be great: distension of the cavity by a positive pressure of air acccunts for most of the size of the cavity in these cases. Nevertheless, to effect complete closure of a cavity a more powerful, prggressive contractive force within the cavity walls would be required. Fibrous tissue is recognised as the type of connective tissue which is capable of progressive contraction. The contraction of scar tissue is due to the fibrous tissue element present.

The belief that the cavity walls are often capable of self-retraction is well established. It is only within recent years that the importance of this process as a mechanism of cavity shrinkage has come to be shared with that of closure of the draining bronchus. It seems however that the latter process is coming to be recognised as the more important of the two methods of cavity closure. Originally Pinner (51) believed that fibrosis played the leading part in cavity behaviour. Scar shrinkage of concentric fibres in the cavity wall tended to close a cavity, whilst retraction of fibrous tissue attached in a radial manner to the concentric fibres tended to pull the cavity open. Pinner was supported in this view by Fischel (28), amongst others. Pinner never found any reason to abandon this conception, although he came to accept the importance of the bronchus in cavity behaviour. Coryllos and Goldberg (29) challenge this conception on the grounds that histologically such fibres have not been differentiated. But Pinner (53) has been able to demonstrate both types of fibrous tissue structure. The impression gained from the most recent published writings of Pinner (53), however, is that the radiating fibrous tissue, described as usually following the perivascular, peribronchial and septal spaces, is no longer regarded by this authority as playing such an important part in cavity opening.

Coryllos and Ornstein (20) (1939) believed that the cavity walls played no active part in the closure of cavities. Cavity closure, they maintained, was entirely the result of closure of the bronchus followed by absorption of the contained air. The walls of the cavity played a passive role, being subject to the changing conditions which followed closure or opening of the bronchus. With a patent bronchus tubercle bacilli thrived under the aerobic conditions within the cavity and tuberculin was produced. As a result of the increased tissue sensitivity and perifocal reaction which occurred in response to the tuberculin, the cavity walls became thickened. If the bronchus became closed the anaerobic conditions within the cavity led to a reversal of these processes and the walls of the cavity virtually disappeared. As will be shown later, this particular hypothesis appears to have been superceded in the later writings of Coryllos (29). Coryllos and Ornstein stated that usually cavities healed with no trace of a scar; that if Pinner's theory of fibrotic retraction were true, a scar would always be formed and so would crumpling of the internal fibres of a cavity as a result of the concentric contraction of the peripheral fibres: furthermore, they added,

cavity closure would be gradual and never sudden; the cavity would never open again; and old thick-walled cavities should close better than thin-walled cavities, whereas the reverse is true.

Pinner (53) concedes that the early cavities with little in the way of a connective tissue wall will rapidly close as a result of bronchial occlusion and heal leaving a minimal scar. The thicker-walled, slower shrinking cavities usually leave a bigger, star-like scar. Ornstein, writing with Ulmar (29) (1946) admits the fact that Tchertkoff in their Department, has repeatedly found a linear scar marking the site of a former cavity of type III (bronchial closure type) of their classification (page 68). Pinner points out, however, that post-mortem observations on healed cavities are rare. Perhaps the answer to the problem is that few cavities heal without residual inspissated contents, the exception being the early cavity with practically no differentiated wall, which so frequently closes rapidly by the bronchial occlusion method. Most authorities are in agreement that these cavities leave little in the way of a scar. As has already been shown (page 103) cavity healing with inspissation of caseous contents appears to be the most frequent form of healing, whilst scar healing - except in the case of early healing - is regarded as being uncommon.

One of the cases "(Case 1)" in the post-mortem studies of Loesch (40) demonstrates the crumpling within the cavity wall which Coryllos denied. There can be no doubt regarding the cases of Loesch that there was shrinkage of the cavity but it is very difficult to say how much of that shrinkage was purely elastic collapse as a result of the pneumothorax, how much was the final forceful shrinkage of the cavity following air absorption due to the ultimate closure of the draining bronchus, and how much was due to fibrous tissue shrinkage of the capsule. In this respect it would be unwise to risk reading too much into the study of the histology as reported. Certain points however invite comment: In Case 3 (fig.11), there is corrugation throughout all thicknesses of the capsule in one area. This would suggest shrinkage by means other than self-retraction of the cavity walls. In Case I, on the other hand, corrugation is mainly of the inner cavity wall which is surrounded outside by hyaline connective tissue. But this cannot be accepted as evidence of fibrous tissue retraction of the outer wall. Loesch interprets the histological appearances as suggesting gradual replacement of the cavity wall by "fibrotic tissue" in the course of closure. This would suggest

that a capsule had been formed around a cavity which had already shrunk as a result of factors such as elasticity and internal absorption of air. Loesch in fact attributes shrinkage of these cavities to their being incorporated in the process of retraction and collapse of the lung, which is due to its elasticity.

It can be said that there is nothing from a study of these cases contradictory to the hypothesis of fibrous tissue contraction of the capsule. The evidence suggests that such a process did contribute towards the closure of the bronchial connections. If that was indeed so, it seems reasonable to suppose that such a process took some part at least in the gradual shrinkage of the cavities under consideration.

Apart from massive fibrosis in a lung incorporating a cavity and so closing or shrinking that cavity, the question remains as to what extent the cavity walls are capable of independent gradual fibrous tissue shrinkage.

There are two sets of instances which strongly advocate that some cavities can close by scar tissue retraction in their walls: They are the pathological findings of Pinner with reference to two cases; and the results reported on open cavernostomy.

Pinner (52) described two post-mortem cases. In Case 1 a scar had formed in place of a cavity. A histologically normal bronchus ran right up to the scar. There were several distorted and dilated small bronchi to be seen within the fibrous tissue of the scar. It was difficult to assess whether the bronchus closed first and so closed the cavity or whether cavity occlusion caused closure of the bronchus. Pinner was inclined to the latter alternative on account of a patent lumen being found within the fibrous tissue. In Case 11, a small shrunken cavity 0.5 cm diameter, was found within a scar similar to the one found in Case 1. The broncho - cavitary junction was still patent, a bronchus being followed into this cavity. Histologically the cavity and scar showed striking resemblance to the findings in Case 1. The cavity wall was only hyaline scar tissue with no granulation tissue. Smears from the cavity contents contained tubercle bacilli. Thus almost complete anatomical healing of the cavity had occurred in the presence of a patent bronchus. Had the patient lived longer complete healing leaving a scar might have resulted. Pinner concluded that the same process of fibrotic obliteration of the cavity in the presence of a patent bronchus might have taken place

in Case I. In the first case, as the author admits, it is only possible to speculate as to what might have happened. To have formed a true picture of the early history of a scar, such as in this case, would have been a problem which even the serial histological section method of Loesch (p. 82) might have failed to elucidate. The second case, on the other hand, is much more convincing.

A number of authorities (16) (46) (39) (56) (58) have reported successful results from opening a cavity to the atmosphere through the chest wall, sometimes accompanied by packing the cavity with gauze. Certain factors, however, have to be taken into consideration when assessing the results in these cases from the point of view of the question in hand at the moment. The success of the cavernostomy was judged in individual cases by evidence such as the radiological disappearance of the cavity, conversion of sputum or sinus discharge to T.B. negative, or the healing of the sinus. No pathological reports have been available on any of these cases subsequently, to see the extent or exact mechanism of cavity closure. Furthermore, in most cases the elastic retraction of the cavity walls and pericavitary tissues such as Monaldi describes (page 137), would have contributed to some extent

towards the shrinkage of the cavity; and in some of the cases in which the sinus had closed before the cavity, the latter might have finally been occluded by air absorption if the bronchial outlet had also closed. When these have all been taken into consideration the fact remains that the evidences from these cases of cavernostomy strongly suggest the operation of scar tissue retraction in the cavity walls.

In view of the objections raised by Coryllos and Ornstein (page 139) and the inconclusive nature of the evidence available to substantiate Pinner's hypothesis of scar tissue contraction of cavity walls, it becomes necessary to examine closely the histogenesis of fibrous tissue as related to the cavity walls.

The fact that connective tissue forms a part of the walls of a number of cavities needs no substantiating. Histologically this has been described by Pinner (53), Loesch (40), Auerbach and Green (4), Kayne, Pagel and O'Shaughnessy (34), and numerous others. Connective tissue is one of the later developments of the proliferative reaction, and proliferation is accepted as one of the processes at work in the formation of cavity walls. Frequently specific reference is made to fibrous connective tissue in or around the walls of

a cavity but many authorities are vague in their reference to this subject. Ornstein and Ulmar in their classification of cavities (page 68) refer to "cicatricial" walls in type (IV) cavities and a "thick cirrhotic wall" when referring to a cavity of type V. Jaffé (29) refers to the presence of "collagenous fibrils" in a cavity wall. But even in the minds of those who refer to "fibrotic" cavities there appears to be some looseness of terminology, using the terms "fibrosis" when "connective tissue" should be applied. According to Schafer (64), fibrous tissue is but one form of connective tissue, the others being areolar, elastic, reticular, adipose and also cartilage and bone. As already pointed out, fibrous tissue alone of all the types of connective tissue, is capable of self-retraction. Other forms of connective tissue are either resilient or, as is mainly the case, impart differing degrees of rigidity to the cavity wall.

Kayne, Pagel and O'Shaughnessy (34) state that "All sorts of connective tissue fibres can be formed in tuberculous tissue: "precollagen" reticulum fibres, collagen fibres and elastic fibres". Jaffé, in describing the histology of the wall of a cavity mentions "collagenous fibrils" but, like Kayne et al, makes no mention of fibrous tissue. Yet both these authorities describe the developement of fibrous tissue

in connection with the epithelioid cells.

If all cavities which persisted in cases of apparently healing tuberculosis had walls composed mainly of fibrous scar tissue it would be expected that progressive shrinkage of cavities would commonly occur with the passage of time: such a phenomenon would be part of the healing process of tuberculosis. But this is not the case.

The impression is gained from the classification he gives, that Pinner considers fibrous tissue takes a definite part in the composition of the walls of cavities of the second and third categories, but that with increasing age of the cavity wall there is a tendency for the fibrous tissue to become hyalinized. When this happens the property of contraction of the walls, which leads to shrinkage, gives place to rigidity, which may develop to such an extent as to defy the most effective therapeutic collapse measures.

Coryllos (29) believes that the chief cause of fibrosis is anoxaemia of the tissue which is frequently due to ischaemia. Because impaired blood circulation accompanies atelectasis, this latter condition often leads to the deposition of fibrous tissue round about a

cavity. Stivelman (68) similarly believes that deprivation of oxygen by this means results in "fibrosis" of the lung parenchyma.

According to Pinner (53), proliferation, or production as he prefers to call it, in pulmonary tuberculosis gives rise to two processes of fibrous tissue formation in and around a cavity walls. One process concerns the epithelioid cells of the tubercles. These tubercles may be in the cavity walls. Rich (57) describes caseous foci being encapsulated by sheets of epithelioid cells and tubercles. A cavity might originate from a tubercle or collection of tubercles. The second fibrous tissue process is the normal connective tissue response to chronic inflammation where granulation is being formed. This takes the form of fibrous tissue encapsulation of the focus of infection.

The histogenic origin of fibrous tissue is still a matter of uncertainity. There seems, however, to be some measure of agreement amongst authorities (Jaffé (29), Kayne, Pagel, O'Shaughnessy (34) and Rich (57)) that the epithelioid cells, or more particularly the reticulum which forms between the cells, is in some way connected with fibrous tissue formation. Jaffé believes that the greater part of this reticulum is derived from the epithelioid cells and that the reticulum is later replaced by "fibrillar connective

tissue" which has a tendency to undergo hyalinization. Kayne, Pagel and O'Shaughnessy, on the other hand, consider that the fibrous tissue is not directly descended from the epithelioid cells but that the "undifferentiated area in the immediate neighbourhood of the cells, apparently under their influence, may be converted into fibres". These "fibres" do not necessarily constitute fibrous tissue fibres but presumably connective tissue fibres in general. As previously remarked, these authorities make no specific reference to fibrous tissue in this connection. Like Jaffé, they refer to the formation of collagenous fibres in tuberculous tissue but give no special place to fibrous tissue. Rich likewise is unable to provide any further solution to the problem of the origin of fibrous tissue. Unlike Kayne et al, however, this authority at least gives the satisfaction of differentiating this form of connective tissue from the various other forms. Rich considers that the epithelioid cells are mononuclear phagocytes which have altered primarily as a result of the ingestion of the tubercle bacillus. The reticulum which forms in the tubercle between the epithelioid cells is gradually transformed into collagen fibres. If the bacilli within the tubercle cease to multiply the tubercle

becomes "static", in which case the reticulum becomes more and more dense and the tubercle becomes more and more "fibrous", until finally it is converted into a "nodule of hyalinized fibrous tissue." Rich declares: "The origin of fibrous tissue of the tubercle is not yet clear", and adds that it is at present no more than an assumption to postulate, as do some authorities, that "fibroblasts wander into the nodule and produce fibrils". Cells can be found in older tubercles, he states, which have the appearance of fibroblasts but which in reality might only be mononuclear phagocytes. Rich concludes: "If it could be established that mononuclear phagocytes can really become fibre producing connective tissue the matter would be of no little importance, not only for the understanding of tuberculous lesions, but from the standpoint of the mechanism of repair in general."

With reference to the origin of the second fibrous tissue process, namely that resulting from granulation tissue formation, there appears to be no more concise knowledge than there is regarding the origin of the fibrous tissue in connection with the epithelioid cells. As Rich points out, tuberculous exudates become organised into connective tissue in just the same way as exudates which are the result of

any other injurious agent, except that they may contain tubercles, giant cells or foci of caseation. In this respect Rich describes how caseous material might become encapsulated either by connective tissue which has formed through the ordinary method of organized repair, or by sheets of tubercles and epithelioid cells which eventually become "fibrous". Pinner (53) draws attention to the apparent paradox that exudative processes may cause a greater degree of reactive fibrosis than is promoted in a proliferation lesion. Caseous matter within an incipient cavity or blocked within a cavity does in fact appear to promote thicker encapsulation than is the case with a cavity which only contains air. Pinner (53) concludes that "the histogenic origin of fibrosis is sub judice:" he adds that it has been described as originating from preformed normal fibroblasts; again as a developement from the large mononuclear exudate cells; whilst there is experimental evidence in support of an acellular origin in inflammatory exudates.

Thus it would appear that as yet the origin and subsequent history of fibrous tissue in the tuberculous reaction is still a matter of uncertainty,

particularly with reference to the amount of such fibrous tissue likely to be formed under various conditions and its survival period. One is forced to admit, as a result of the foregoing enquiry, that the armoury from which to combat the sweeping criticisms of Coryllos and his equally forthright substitutions (p.138-9) is ill equipped with the necessary histogenic data.

It is necessary to take a broad view when considering the so-called walls of a cavity and the question of their ability to contribute towards or resist cavity closure. The cavity walls consist of lung parenchyma which has undergone histological changes in response to the activity of the tubercle bacillus. These responses vary; either necrosis, exudation or proliferation may predominate, or one or more of these processes may be superimposed upon or intermingled with one or more of the others. The cavity wall may at one time be the site of a localised pneumonic consolidation when it will appear radiologically as a wide ring of density. At another time the same cavity may appear surrounded by a thin ring of atelectatic air cells, the pneumonic process

having subsided and the cavity having become inflated due to a check valve mechanism within the draining bronchus. These features are well demonstrated in the cases presented at the end of this thesis. With such cavities there can be no stable conception of a cavity wall at one particular stage in its life history. Any connective tissue element in the walls, on the other hand - be it fibrous, elastic or hyaline tissue - will naturally remain relatively constant: a proportion of it will be permanent. A sudden increase or decrease in the thickness and density of the cavity wall as seen radiologically must be ascribed to changes in the pneumonic inflammation or the degree of atelectasis present, which alone are capable of rapid fluctuation.

These pathological changes must to a large extent be the result of biological changes within the cavity - degrees of activity of the tubercle bacilli they contain, in other words. These in turn are materially effected by the state of aeration of the cavity, which again is dependent upon the state of the draining bronchus. To this extent one is prepared to accept Coryllos' hypothesis regarding the factors responsible for the pathology of the cavity

walls. In the light of the indisputable fact, however, that scars, small or great, are frequently left at the site of a former cavity it is justifiable to accept that there is also a more permanent structure frequently to be found in cavity walls. These scars consist largely of hyaline tissue with a smaller, and perhaps younger, fibrous tissue element present. It has already been shown that connective tissue is present in many cavity walls. Whether or not Coryllos included connective tissue under the heading of the perifocal reaction to tuberculin which took place in a cavity walls, it is not understood. If this was so, the implication must be that such connective tissue did not, in Coryllos' estimation, last permanently, but was absorbed when the cavity finally healed. An explanation would appear to be offered when it is realised that Coryllos did not recognise that there was such a thing as a cavity wall, but regarded the so called wall in the strict sense of its being lung parenchyma. When he refers later (29) (1946) to fibrous tissue formation in the tissues surrounding a cavity as a result of atelectasis it may be he is referring to what Pinner would call fibrous tissue in a cavity walls. In these later writings

Coryllos appears to be silent with regard to his hypothesis of the tuberculin reaction within the cavity walls and refers instead to atelectasis, following the pathological occlusion of bronchi and bronchioles, which in turn leads to fibrous tissue formation. This explanation, however, still leaves unsolved Coryllos' belief that cavities heal without a scar.

Except in the case of Pinner, there is a remarkable dearth of direct reference by authorities to the histological demonstration of fibrous tissue in cavity walls. This becomes all the more so if the statement of Rich is to be accepted that mononuclear phagocytes may resemble fibroblasts.

To the poorly supported evidence of Pinner that fibrous tissue is found to a considerable extent in the walls of cavities must be added the evidence which is provided by cavities which have healed as a result of cavernostomy, where the internal pressure has remained atmospheric. These latter instances strongly suggest that there exists some measure at least of self-retraction within the walls of cavities. Such self-retraction, as has been shown, can only be due to fibrous tissue. To what extent this self-retraction

occurs must vary, depending partly upon the amount of fibrous tissue laid down. As a means of cavity closure such a process must for the most part be overshadowed by the more effective static intracavitary force produced by closure of the bronchus, or by the filling of the cavity lumen with caseous matter.

Connective tissue will impart a certain amount of rigidity to the cavity walls. This may not normally amount to very much but will increase with the age and thickness of the cavity walls. Saley (60) writing in 1931 considered that the pathology of the cavity wall was important from the point of view of the collapsibility of the cavity and that collapse therapy should be started before the cavity had had a chance to form a fibrotic wall. Fischel (28) writing two years later expressed belief in a rigid cavity where excess of fibrous tissue in the cavity walls caused rigidity as opposed to elasticity. Monaldi (43) (44) on the other hand objects to such a conception on the grounds that a homogeneous wall of connective tissue cannot, generally speaking, form round a cavity. The resilience and retractive properties of some cavities might be reduced, he maintains, as a result of the pathological processes

around their walls and involving the parenchyma, which might include the fracturing of elastic fibres. In addition pathological deposits are to be found on the cavity walls. These can be removed by appropriate treatment, when the cavity will become elastic once more.

It is unlikely that a thick-walled cavity would remain patent solely on account of its rigidity. There would probably be some fibrous tissue present which, given the time and opportunity, would result in progressive contraction of the cavity walls.

To summarize the conclusions thus reached it may be stated that the origin of fibrous tissue in a cavity walls remains uncertain but it appears to be connected with the epithelioid cells of the proliferative tuberculous reaction. In addition fibrous tissue is produced as part of a normal response to chronic inflammation. As suggested by Coryllos, anoxaemia, mainly in the form of ischaemia, has the effect of promoting fibrous tissue production. For practical purposes and for want of a better working hypothesis Pinner's implications may be accepted, that in young cavities with thin fibrous tissue walls self-retraction may occur if conditions permit; whereas with increasing age of the cavity and the gradual transition of fibrous tissue to hyalinized fibres, rigidity takes the place of contraction.

SECTION IV.

CLASSIFICATION OF PERSISTENT CAVITIES.

Cavities which persist in spite of the normal methods of collapse treatment having been tried, can be classified according to the factors which are causing them to persist. In the light of the fore-:going deductions these factors fall into three categories:

I. Extracavitary forces which, by acting eccentrically upon the cavity walls, prevent them from coming together and so closing the space. In this class are adhesions of the lung to the walls of the bony thorax.

II. Endocavitary forces produced by gases at a pressure greater than that of the atmosphere which distend the cavity and so prevent its walls from coming together.

III. Factors which cause the cavity walls them-:selves to resist closure because of rigidity.

As has been maintained from the outset, consideration is not being given to such conditions as pregressive excavation of the lung by continued exudation and necrosis, nor to cavities which persist because collapse measures, which are regarded as effective in the majority of cases, have not been tried. It will

generally be found that a cavity which persists in spite of an apparently effective artificial pneumothorax or an apparently adequate thoracoplasty, for example, will be remaining patent because of factors which fall into one or other of the above categories; or, as is more often the case, because of a combination of such factors.

The days of persisting with an ineffective artificial pneumothorax are now fortunately passing. Nevertheless many an apparently effective artificial penumothorax will be continued with disappointing results if assessed by radiological appearances only. Thoracos-:copy should be performed as a routine procedure a few weeks after induction of an artificial pneumothorax. An anterior or posterior adhesion may not be apparent even in a lateral skiagram.

Perhaps one of the most troublesome and frequent sites for persistent cavities is in the upper zone close to the mediastinum. An artificial pneumothorax fails to relax the apex towards the hilum; a phrenic paralysis and pneumoperitoneum is probably rarely effective, unless the cavity is situated more peripherally, owing to the inability of this procedure to lift the root of the lung sufficiently upwards towards the fixed apex. With a thoracoplasty it is often impossible to carry out an effective apical strip from the mediastinum; the cavity is left suspended between the relatively fixed mediastinal portion of the apex and the root of the lung, collapse measures having produced lateral compression only.

The factors responsible for cavity distension are to be found in the draining bronchus. Distended cavities are, in varying degrees, positive pressure cavities. Their origin may be ascribed to any of the causes enumerated in the section devoted to intermittent closure of the bronchus. The underlying cause is most frequently endobronchial disease.

Old cavities with rigid walls composed largely of hyalinized connective tissue may resist the apparently most successful surgical collapse of the lung. Even with a closed bronchus and high negative intracavitary tension, such a cavity may refuse to close. In the course of time, however, such a cavity would be likely to undergo slow, progressive shrinkage of its lumen due to the fibrous tissue element in its walls and slow absorp-:tion of air, provided there was no interfering mechanical factor.

Subpleural cavities are particularly prone to afford trouble on account of their tendency to persist in spite of collapse therapy. This is due to a combination of factors. Compensatory emphysema of the surrounding air spaces acts as a factor which helps to fill in a cavity space. This factor does not apply to the same extent in the case of subpleural cavities. Even with a persistently negative intracavitary pressure the lung subjacent to the pleura might be incapable of filling up the gap unless the negative intracavitary tension was high. As the weight of the lung is largely

supported by the negative intrapleural pressure, it is probably more correct to regard the cavity space as being occupied by the surrounding spongey tissues through compensatory emphysema, rather than to regard the weight of the surrounding lung as causing compression. Subpleural cavities persist for the same reasons that any cavity may persist. If they are more troublesome than many others this is partly due to the factor just described but also, being peripheral cavities they are drained by small bronchi at the end of the bronchial passage - conditions which are liable to obstruction of the inflation check valve variety. Subpleural cavities include cavities situated at the apex of the lower lobe or close to the paravertebral gutter, both of which have a reputation for resisting attempts at closure. By keeping out of the way in the paravertebral gutter, a segment of lung in this situation containing a cavity might escape sufficient collapse from a thoracoplasty, with consequent failure of the cavity to close.

SECTION V.

DIAGNOSIS AND TREATMENT.

D I A G N O S I S

Before a further attempt is made to close the persisting cavity, it is essential to arrive, as far as possible, at some conclusion regarding which of the factors, or combination of factors, mentioned in the previous section is responsible for this condition. A careful reassessment of the original nature of the pulmonary lesion based upon the history, signs, symptoms and X-ray findings will be necessary. In some cases, features responsible for the failure on the part of the cavity to close will be readily evident. Pleural adhesions may be noted radiologically; the apex may be adherent to the superior mediastinum; or signs of basal adhesions may be apparent in peaking of the diaphragm. On the other hand it might be necessary to resort to thora-:coscopy, perhaps for a second or third time, in the case of a pneumothorax in order to determine the presence and nature of adhesions.

It has already been indicated that tracheo-bronchitis is a frequent underlying cause of persistent cavitation. Symptomatically there is frequently a history of an irritating cough accompanied, maybe, by a slight blood-stained sputum containing tubercle bacilli. Rafferty (55), amongst others, draws attention to the

fan-shaped radiological shadow in tracheo-bronchial disease stretching from the hilum to the cavity area. There may simply be prominence of one or more bronchial shadows on the X-ray film, as shown in two of the cases (Cases III and IV) studied in this thesis. Bronchoscopy almost always provides conclusive evidence of endo-:bronchial disease for, as Lemoine and Langeard (38) point out, the state of activity of the parenchyma, or a cavity, tends to have its reflection in the appearances of the mucosa of the bronchi which are within the range of a bronchoscope. If the larger bronchi are affected it is unlikely that the smaller, more peripherally situa-:ted, branches are not involved. From this, however, it can only be deduced that a positive finding through the bronchoscope, is of value. If the bronchi appear normal it cannot be concluded that the smaller bronchi are not affected. Referring to the treatment of cavities by collapse therapy Vineberg and Kunstler (71) maintain that the state of the draining bronchus must be assessed by a study of the intracavitary pressures because with the bronchoscope it is not possible to see far enough; consequently bronchoscopic assessment is unreliable.

With the increasing recognition being given to tracheo-bronchial disease there has developed an increas-:ing caution on the part of tuberculosis clinicians towards embarking upon major collapse measures, especially artificial pneumothorax, if there are signs of tracheo-bronchitis being present. The cautionary attitude in

the minds of clinicians as to when and when not to induce an artificial pneumothorax in the presence of different degrees of tracheo-bronchitis, varies. The majority are probably agreed that such a method of treat-:ment is contraindicated when there are signs of stenosis of the medium and larger sized bronchi. The picture of massive lobar or segmental collapse of the lung, or of ballooning of a cavity, has now become fully recognised as one of the accompaniments of major collapse therapy in the presence of endobronchial disease.

The radiological appearance of a tension cavity is as typical as is its behaviour. The spherical outline, often surrounded by a thin ring of atelectatic air cells, with one or more linear bronchial shadows radiating towards the hilum, clearly denotes a tension cavity. Such a picture is generally accompanied by a history of either sudden ballooning of the cavity or of fluctuations in size from day to day, or week to week, and often with a changing fluid level in the cavity being visible on the screen or series of films.

In a similar manner the history and clinical picture accompanied by the X-ray appearances of the cavity, and the behaviour and appearance of the lesion in which it is centred, will frequently provide evidence regarding the nature of the cavity wall: this might be almost completely absent (Pinner type I); it might present as a very thin ring of fibrous tissue (Pinner type II); or it might appear as a thick, unchanging annular shadow such as is found in chronic fibroud phthisis (Pinner Type III).

Lateral and tomograph films will frequently be of assistance not only in helping to establish the nature and position of the cavity, but in determining the course of future treatment, especially when pulmonary resection is being considered.

T R E A T M E N T

The methods of treating persistent cavities are legion. Their very numbers bear witness to the difficulty which is often met in inducing some cavities to close, and indicate that no entirely satisfactory method, short of pulmonary resection, has yet been found. Naturally enough it will be desirable to spare the patient major surgical procedures if at all possible but this attitude should not give place to a long and disappointing series of minor surgical measures. The patient has very likely already been subjected to one or two changes of treatment.

Treatment will be directed towards removing whichever of the factors or combination of factors described in Section IV is responsible for keeping the cavity open. As rigidity of the cavity wall must seldom be the sole cause of patency of a cavity, given time for slow scar shrinkage to occur, treatment will be directed mainly against extracavitary and endocavitary forces acting upon the cavity.

R E S T

Rest in bed is an essential in the treatment

of a persistent cavity. The advantages secured by this means, both local and systemic, help to throw the balance in favour of the proliferative process and against the exudative and necrotic processes; the result being that fibrosis rather than necrosis will tend to occur in the cavity walls. Similarly rest promotes healing of the bronchial disease with reduction of the mucosal swelling and secretions. At the same time the amount of cough will be reduced and respirations will be quiet. Under such conditions forced inspirations will be minimal with the result that inflation of a positive pressure cavity will likewise be reduced to a minimum.

There comes a stage, however, in the treatment of stubborn cavities when no useful purpose is served by imposing too strict rest on the patient. Reliance will have to be placed upon the more direct surgical means which are applicable to that particular case. If major surgery is going to be required an increase in the patient's freedom will probably do more good as a preliminary to operation, because of the mental stimulus produced, than will unwarranted caution.

WHERE THE CAUSES ARE EXTRACAVITARY.

When a cavity is prevented from closing by extracavitary forces, treatment will be directed towards promoting further relaxation of the part of the lung con-:cerned. Hitherto relaxation might have been brought about by partial collapse of one of the walls of the

thoracic cage - by removal of ribs or raising the diaphragm. That having been the case, further collapse may be produced by removal of more ribs or parts of ribs, in the case of a thoracoplasty; or by supplementing a phrenic paralysis with a pneumoperitoneum. If, on the other hand, collapse has been produced by separating the visceral pleura from the walls of the thorax by means of an artificial pneumothorax, further collapse will probably only be possible after further division of adhesions: this may or may not be possible.
Recently enucleation has made it possible for a number of adhesions containing lung tissue to be dealt with safely. In Sweden, Hedvall (72), amongst others, performs "open pulmolysis" on cases in which thoracoscopy has shown adhesions which are unsuitable for cauterization on account of their size. The operation is an enucleation on a large scale. Having resected 6" of rib, the pleural cavity is entered and the adherent area is encircled by an incision of the parietal pleura made from within the pleural space. The lung is thus freed from the chest-wall. The edges of the raw surface on the freed lung, composed of adherent visceral and parietal pleura, are brought together and sutured, thus invaginating the walls of the cavity lying underneath.
At Uppsala, by 1947, Hedvall had treated 66 cases by this method with good results in 70% of cases. Cavities had been present in 50 of these cases and in 43 these disappeared. Positive sputum had been found in 51 and 40 of these became T.B. negative; of the remaining

11 whose sputum continued to be positive, 5 were considered to derive the positive sputum from the contralateral lung. Complications consisted of mixed infection empyema in 12 and tuberculous empyema in 5. In the mixed infection empyema the incidence was reduced from 8 in the first 19 cases, to 4 in the subsequent 47 cases, all of which latter were treated with penicillin. Of these 4 mixed infection cases 3 were free from infection after one week. Of the 5 tuberculous empyema cases 2 had had the empyema before operation, and by the time of the report 4 of the 5 had cleared. There were 7 deaths in the 66 cases: 3 of these died of empyema; the other 4 cases were late deaths, some of them having been poor risk cases. Open pulmolysis is considered to offer a form of treatment intermediate between cauterization of adhesions and the more major operation of thoracoplasty, which latter opera-:tion would probably have been performed in this country. This operation would appear to be a most promising form of treatment if results continue to be as good as those recorded by Hedvall.

Only too often, however, the apex of the lung is held by adhesions to the superior mediastinum, and it is not often a thoracic surgeon can risk attempting to strip the pleura internally in this region, and dense adhesions may prevent an extrapleural strip also.

Sometimes when the adhesions cannot be cut, a satisfactory result may be obtained upon taking the strain off the adhesions by relaxation from one or other

end. Either the lung can be moved towards the attachment of the adhesion to the thoracic wall, as when phrenic paralysis is carried out for an apical adhesion; or one of the walls of the hemithorax can be moved towards the attachment of the adhesions to the lung, as when phrenic paralysis is performed where there are diaphragmatic adhesions to the base of the lung. The same principle is observed when a thoracoplasty is carried out on account of adhesions of the lung to the chest wall. Extrapleural pneumothorax, an operation favoured in Scandinavia (72) and perhaps gaining popularity in this country, achieves its object through a similar mechanism; creating a relatively localised relaxation of the extracavitary forces and circumventing internal pleural adhesion. From whichever end of the adhesions the strain is relieved the result is a reduction in the extracavitary forces holding the cavity open. This force having been reduced, the cavity might now be able to close.

Thus, should a further attempt to divide the adhesions in a pneumothorax be considered futile or injudicious, the required relaxation can often be obtained by supplementing the collapse already obtained with a phrenic paralysis, with or without the addition of a pneumoperitoneum. This is particularly so with certain upper zone cavities which are being prevented from closing by apical adhesions, or in basal cavitation with diaphragmatic adhesions. In the former case it is often found that provided the edge of the apex of

collapsed lung does not lie lateral to the inner third of the clavicle when viewed on the ordinary postero-anterior skiagram, a phrenic paralysis, especially when assisted by a pneumoperitoneum, will permit the necessary medial or hilarwards relaxation of the apex to occur which will close the cavity.

In cases with bilateral disease, collapse therapy upon one side is not infrequently found to benefit the other lung: sometimes this may even lead to closing a cavity. In such cases the shift of the media-:stinum has produced relaxation of the contralateral lung from a medial direction.

In conclusion it may be said that whilst the judicious combination of artificial pneumothorax with phrenic paralysis - and often pneumoperitoneum - has its place in the armamentarium of the treatment of cavities, the results in the cases of those cavities which have failed to close with the former method of treatment alone are often disappointing, and much time can be lost and tedium imposed upon the patient (as in Cases II and III presented at the end of the thesis) by persisting with these means for too long a time. According to Walsh (72), in Scandinavia when artificial pneumothorax has failed, the next step is major surgery: phrenic paralysis is generally regarded as being so much waste of time and is hardly ever used; whilst pneumo-:peritoneum is never performed. In this country there is a growing tendency to abandon at an early date any artificial pneumothorax which does not give the

appearance of being satisfactory, although the supplement of a phrenic paralysis with or without pneumoperitoneum is still regarded favourably. Whether the pendulum is beginning to swing too far away from pneumothorax treatment in the direction of thoracoplasty only time will show.

An artificial pneumothorax has the advantage of producing concentric collapse of the lung towards the hilum, which is mainly selective to the diseased portion provided there are no adhesions. Other methods of treatment which involve collapsing one of the walls of the thorax will be effective according to the rôle played by that wall in the respiratory movements of the thorax. The modern Semb thoracoplasty also produces concentric relaxation of the lung but principally affects that part which underlies the ribs or parts of ribs which have been removed. A thoracoplasty, however, like a pneumothorax, may fail to provide the necessary relaxation when superior mediastinal adhesions interfere with the apical stripping. It is because of its per-:manence, the degree of immobility, and the satisfactory degree of collapse generally provided, that the modern thoracoplasty operation has found such a secure place in the treatment of cavities. Coryllos (29) believes another virtue of this operation to be the permanent atelectasis and ischaemia produced, with resultant fibrosis, which is especially marked in the diseased area. It can readily be understood that the production of fibrous tissue will contribute towards the occlusion

of both the draining bronchus and the cavity itself. In phrenic paralysis, if reference is made to figure I and the section dealing with the respiratory movement of the lung, it will be seen that the lung is relaxed in a vertical direction, especially in its posterior segments: the greatest effect will be produced in the lower half of the lung. It is probable however that the relative difference between the amount of anterior and the amount of posterior collapse will be reduced in the case of a pneumoperitoneum owing to the greater general rise of the diaphragm.

WHERE THE CAUSES ARE ENDOCAVITARY.

In spite of having provided adequate pulmonary relaxation a cavity will often fail to close. In these cases the cause is nearly always that the cavity is being kept patent by forces acting from within, and steps must be taken to deal with a positive static intracavitary pressure - in other words an inflated cavity. The most frequent cause of a raised intra-:cavitary pressure is endobronchial disease which has created an intermittent bronchial occlusion, the effect being that of a check valve.

The mode of treatment when a check valve is at work may be approached from various angles:

I. Treatment may be directed towards the valve; the purpose being to convert an intermittent bronchial occlusion into either (a) an open or (b) a closed

bronchus. If these means fail or are for any reason unsuitable then the next method of approach may be tried.

II. Treatment may be carried out in spite of the valve; that is by ignoring the site of the valve mechanism and seeking to create the conditions of an open or closed bronchus by other methods. The methods available are to create (a) an atmospheric or negative pressure cavity by a transthoracic approach or (b) a closed bronchus by occlusion at some site other than the valve.

I. TREATMENT OF THE VALVE BY CONVERSION TO (a) AN OPEN BRONCHUS.

The principal agents responsible for intermittent bronchial occlusion are mucosal swelling, fibrous stricture and granulation tissue, which cause narrowing of the bronchial lumen. These are usually combined with secretions, caseous matter and necrotic debris. All these agents are the direct result of tuberculous bronchial disease or of the activity of the tuberculous disease within the cavity walls. Thus it will be seen that any treatment directed towards producing quiescence of the tuberculous bronchitis or of the tuberculous process in and around the cavity walls will contribute materially towards abolishing the intermittent occlusion of the bronchus. Stenosis of the bronchial lumen will be relieved when the oedema subsides and granulation tissue heals: there will be less secretions, caseous matter and debris when the disease in the cavity

and in the bronchus is less productive. When these improvements have been achieved, the bronchial walls will no longer touch on expiration and solid particles, which will be fewer in number, will be able to pass with less risk of forming a ball valve. Only in the case of the oblique bronchus valve (p.114) can less beneficial results towards the valve mechanism be expected from treatment on these lines. Methods of treatment, however, of a more mechanical nature are also available and have proved effective. Some of the appropriate general, medical and surgical methods of treatment with the aim of creating an open bronchus, will now be described.

(1) Rest and General Measures.

Reference has already been made to the advantages of rest as a means of promoting healing of the tuberculous bronchial disease and of encouraging scar tissue formation in the cavity walls. Rest implies lack of strain, both physical and mental. A well-balanced diet is recommended, supplemented by vitamin A from Codliver Oil, Halibut-liver Oil, or Adexolin tablets.

(11) Ultra-violet Light.

Shipman (65) describes general irradiation with ultra-violet light as a possible beneficial mode of treatment. He records that beneficial effects had been observed prior to 1936 at the Thoracic Surgery Clinic of the University of California Medical School, as a result of treatment by this means. The results of 15 cases treated by Shipman are very inconclusive however:

general rest and artificial pneumothorax might have contributed a good deal to the successes. It is of interest to note, however, that reactions to the lamp were only transitory.

(111) Streptomycin.

It is only within the past six months that streptomycin was first released for the treatment of tracheo-bronchial disease: thus as yet treatment with this antibiotic substance is in the experimental stage. Results obtained so far, however, give rise to the hope that streptomycin may prove to be of the highest value in the treatment of this condition by promoting rapid healing: it may indeed come to be accepted as the treatment of choice before all others. Preliminary treatment of tracheo-bronchial disease with streptomycin may eventually lead to artificial pneumothorax treatment becoming possible where formerly it had been contraindicated.

(1V) Bronchoscopic suction.

Bronchoscopic suction has been advocated mainly by writers on the Continent. Lemoine and Langeard (38) advocate bronchoscopic aspiration combined with local application of adrenalin. By this means they seek to reduce the oedema of the bronchial mucosa and to promote free drainage, having sucked away the secretions clogging the cilia. This process, according to the authors, appears to set in motion a type of

syphon action which encourages the continuous passage of secretions from the cavity towards the larger bronchi. It is important, they state, that suction should be applied as near as possible to the segmental bronchus pf the affected lobe. Adrenalin is applied on a small swab. Treatment is given fortnightly; one to eight sessions generally proving sufficient to result in radiological disappearance of the cavity and conversion of the sputum to T.B. negative. Treatment has mostly been given to patients undergoing pneumothorax treatment who have persistent cavities, or tension cavities which have ballooned out following adhesion section; having been withheld for at least a month in all cases to ensure that the cavity persists: it has only been used by the authors in cases showing no sign of improvement. Out of 38 cases treated by this means since 1939 cavita-:tion has disappeared in 33. The authors claim cure in 32 of these cases, one of whom has remained satisfactory for 8 years, the remainder for varying periods from 1 - 6 years.

Rafferty (55) considers bronchoscopic suction should be tried in cases of tension cavity under pneumo-:thorax, before resorting to the more serious and irreversible forms of collapse therapy. He quotes Meyersburg, Gruber and Lupo as having obtained cavity closure in 3 such cases by this means.

From the report given by Lemoine and Langeard, and others, this method of treatment commends itself as

being effective and based upon sound principles. If treatment is carried out by an expert bronchoscopist the procedure should not be unduly troublesome for the patient. Such a means is preferable to some of the alternative measures which might be required in view of the nature of the condition, always provided no simpler operation is possible or advisable.

(V) Manipulation of the Bronchus.

By this is meant altering the degree or type of collapse of the lung in order to affect the patency of the bronchus draining a cavity. This is really an intermediate classification of treatment as the process might result in opening or completely closing an intermittently closed bronchus.

The extraordinary response of some cavities to an alteration in the degree or method of collapse of the lung strongly suggests that on these occasions intermittent bronchial occlusion has been replaced by total occlusion or else full patency of the bronchial lumen, resulting in cavity closure. By partially re-expanding a collapsed lung in which there is a tension cavity the latter may be observed to close rapidly and remain shut when the pneumothorax collapse is increased once more. Again, by supplementing an artificial pneumothorax with phrenic paralysis, the mechanics of the draining bronchus might be so altered by compression or kinking that complete closure of the bronchus takes place; only a slight movement which affected the bronchial lumen might have been responsible for the complete change.

Steele, Trenis and Laboe (67) described 12 cases in which unexpected cavity closure had occurred upon re-expanding an artificial pneumothorax with a view to thoracoplasty. So impressed were they by these results that, like Bobrowitz (10), they were led to advocate waiting before embarking upon thoracoplasty after pneumothorax to see whether the cavity would close spontaneously as the lung re-expanded. The authors found that cavity closure was more dramatic in cases where a phrenic paralysis had been performed at the time of discontinuing the pneumothorax. In their opinion the phrenic paralysis was not necessarily responsible for cavity closure as most of the cavities had been too large to have been closed so rapidly by this means: in addition, similar results had been observed in some cases without phrenic paralysis. Unfortunately the results of these authorities are somewhat vitiated because the pneumothorax had been established but a short time in some cases (6 weeks in one case; a fortnight or less in 3 other cases): furthermore adhesion section was not carried out. The conclusions to be drawn from this report are that the phrenic paralysis might, to some extent, have been responsible for the closure of these cavities; further, had the artificial pneumothorax been continued longer in some of these cases the cavities might have closed: nevertheless it is very suggestive that rapid cavity closure was being observed as a result of interference with the patency of the draining bronchus, associated with the re-expansion of the lung.

It is difficult to see why letting up a pneumothorax should have closed a cavity if it is maintained that the bronchus must have been occluded by the process: it would be expected that an intermittent occlusion would be converted by this procedure to full patency of the bronchial lumen. It might be asked whether the phrenic paralysis, in these cases, was responsible for kinking the bronchus, or whether the former state of collapse had so altered the pathological anatomy of the bronchus or the surrounding lung that when the latter was re-expanded, occlusion of the lumen took place as a result of kinking or internal blocking of the bronchus. A third, and perhaps more likely, possibility, is that the bronchial lumen opened when the lung re-expanded: the lung tissue surrounding the cavity being in a state of partial collapse - all the greater on account of the recent greater collapse - now closed the cavity which was no longer being maintained by a positive internal pressure. This process, and the shrinking of the recently distended cavity walls, might also close the broncho-cavitary junction; or the original check valve mechanism might not recur: either explanation would serve to account for the fact that such cavities generally remain closed when the pneumothorax collapse is increased once more.

Rafferty (55) also believes that in some cases partial re-expansion of a pneumothorax may result in closure of the cavity. The success of this manoeuvre he attributes to changes at the broncho-cavitary

junction which decrease the efficiency of the valve and allows some air to pass from the cavity.

Shipman (65) describes a case being treated with bilateral artificial pneumothorax. There were two cavities in the right lung and one cavity in the left. The patient's condition was made worse by refills. Upon abandoning both pneumothoraces, all three cavities closed and the patient's condition became good. The explanation given was that tuberculous bronchitis was presumably present. Collapse of the lungs also produced collapse of the draining bronchi creating an inflation check valve. Re-expanding the lungs permitted the bronchi to open up again. It is interesting that the same phenomenon occurred with all three cavities. The author admits that the same explanation might not hold for each of the three cavities.

Bobrowitz (10) reports on his treatment of 5 cases of tension cavity by means of artificial pneumothorax and refers to his experience with 6 other similar cases. Some of the cavities revealed typical tension characteristics after the pneumothorax had been induced; others had this appearance before collapse therapy had been instituted. Of these cases 6 were treated by partially or considerably re-expanding the collapse, to be followed in some instances by increasing the collapse again; the remaining 5 were treated by maintaining a shallow collapse in the presence of pleural adhesions. Bobrowitz believes that pneumothorax changes the direction and angulation of the

bronchus, alters the lumen and modifies the amount of drainage: thus it follows that variations in the amount of pneumothorax collapse can create, accentuate, or remove a bronchial valve mechanism. It is striking the amount of collapse the author permits - up to 85% in one case. A sense of apprehension is felt reading the description of some of the tension cavities to be seen radiologically bulging into the pleural space whilst manoeuvres are carried out with the amount of collapse of the lung. It is not surprising to note the report of one or two pleural effusions. The first case reported gives rise to a considerable amount of speculation: the fact that a turbid pleural effusion developed, T.B. positive on culture, is rather glossed over; added to this is the report that the lung later became markedly collapsed again with disappearance of the cavity for the first time. It is to be wondered whether the cavity ruptured into the pleural space. Although 10 months after discharge the patient's general condition was excellent and the pneumothorax was being continued with a minimum amount of fluid, these facts are not incompatible with such a suggestion. Erwin (27) believes that only 10% of pleural effusions become empyemas, even including occasions when they follow a ruptured cavity; and in some instances the purulent quality will resolve spontan-
:eously. Whilst this doctrine is dangerous if it lessens a sense of respect for the troublesome nature of tubercu-
:lous empyema, nevertheless, if at all true,

it supports the suggestion of cavity rupture in the case in question. The conclusion with regard to the method adopted by Bobrowitz is that it is far too dangerous on account of the eminent risk of tuberculous empyema following rupture of a cavity into the pleural space: consequently this mode of treatment, except in the case of the shallow collapse, is not to be recommended. Although the case described is the only specific empyema, there was more than one other reference made to some degree of pleural effusion during the treatment.

Provided the risks are fully realised a tension cavity which appears during the course of artificial pneumothorax treatment, or sometimes even before the induction, may occasionally be treated successfully by a very shallow pneumothorax collapse. This procedure may at times prove effective when supplemented with phrenic paralysis with or without pneumoperitoneum. To try such a line of treatment when a tension cavity is already evident before the induction must be exceptional however. Many authorities regard pneumo-:thorax treatment in the presence of a tension cavity as contraindicated no matter at what stage the tension cavity appears. Rafferty (55) states that when a tension cavity is found during the course of artificial pneumothorax treatment, generally a change of treatment is indicated, but he adds that in some cases partial re-expansion of the collapse may close the cavity. A

further great drawback to pneumothorax treatment of tension cavities is that the decision to abandon the collapse is fraught with more danger than keeping the collapse constant. Before embarking upon such a course therefore, it would be well to reflect that the responsi-:bility has to be accepted of a return journey which will be more hazardous than the beginning. It is in the process of letting up the pneumothorax collapse that the rupture of the cavity frequently occurs. The reason for this is that upon abandoning the pneumo-:thorax the intrapleural pressure becomes increasingly negative: this results in the intracavity pressure becoming relatively more and more positive until the cavity wall bursts. It will be realised that the risks may be obviated by allowing the lung to re-expand very slowly, giving frequent small refills: the supplement of phrenic paralysis and pneumoperitoneum will help materially. A further safeguard is to ensure that the collapse is never very great: this shortens the return journey. But on each occasion the question will arise, whether the trial is justified because of the risk.

As in the case of collapse therapy, movement of the mediastinum might result in cavity closure by the same principle of alteration in the patency of the draining bronchus. Hershey and Ballinger (31) described a case where a large cavity in the left upper zone was

closed by an artificial pneumothorax on the contralateral lung. Prior to the pneumothorax the cavity had shown a fluid level and there was considerable traction towards the left side on the mediastinum. The right artificial pneumothorax collapse was pushed throughout. The cavity was still closed and sputum T.B. negative more than a year after discharge. Cavity closure they ascribed to kinking of the draining bronchus on the left side causing permanent closure.

I. TREATMENT OF THE VALVE BY CONVERSION TO (b) A CLOSED BRONCHUS.

In the preceding part it was pointed out that changes in the degree and nature of the collapse of a lung brought about by pneumothorax, phrenic paralysis or movement of the mediastinum might result in opening or closing of a bronchus which was formerly intermittently closed.

Thoracoplasty.

Thoracoplasty is but another form of collapse, yet on account of the nature and degree of collapse produced by this means it is more likely to result in occlusion rather than opening of the valved bronchus.

Opinions differ regarding the main mechanical advantage secured by collapse therapy: some authorities hold that it is the relaxation of the parenchyma; others like Coryllos (29) maintain that it is the effect of the general relaxation upon the bronchi to the diseased area,

causing them to become occluded. In this respect Coryllos refers to the fact that the adhesions which most effectively hinder collapse of a cavity are the ones which run postero-superiorly towards the second and third intercostal spaces and the posterior axillary line. Such adhesions must be cut if cavity closure is to be secured. As these adhesions follow the direction of the prolongation of the apical bronchus Coryllos suggests that they act by keeping the bronchus straight and so preventing its collapse or kinking when the apex drops. It is most likely that advantages are secured from both of these mechanical principles.

Coryllos (29) describes two pathological specimens of lungs which had been collapsed by thoraco-:plasty. In one of these lungs a cavity and the draining bronchus had both been closed; in the other lung a cavity was still patent and so was the draining bronchus. Coryllos used this illustration to substantiate his belief that a thoracoplasty must close the draining bronchus if it is to be an effective means of closing a cavity.

The effect of a thoracoplasty on intermittent bronchial occlusion is to complete the collapse of the stenosed bronchus and so produce permanent occlusion. The same effect may be produced by bending or kinking the diseased bronchus. If Coryllos is correct in his belief that this form of surgical collapse is especially

productive of fibrous tissue in the collapsed portion of lung, this factor must contribute a good deal towards the occlusion of the cavity and the draining bronchus. In addition the inspiratory drop in the intracavitary pressure, which is responsible for filling the persistent cavity, will be minimized owing to the relative immobi-:lity of the bony thorax over that area and to the fact that the cavity is surrounded by collapsed and not by expansile lung.

Thoracoplasty, however, does not always close a persistent cavity, especially when the cause of the persistence is a one-way valve of inflation. If the thoracoplasty merely reduces the extracavitary fordes but does not correct the check valve mechanism, it will not close the cavity.

In 119 thoracoplasties, Vineberg and Kunstler (71) (1942) found 12 residual cavities in which, after operation, a highly positive intracavitary pressure was noted. Presumably this was prior to the institution of their transthoracic suction drainage method of treating tension cavities before performing thoraco-:plasty.

Blecher (9) (1945) reported on 549 cases of thoracoplasty performed in the Renströmske Hospital, Göteborg, between 1931 and 1943. There were 51 residual cavities in the series. In 107 cases where

apicolysis had not been carried out there were 14 (13%) residual cavities. Among 442 cases where apicolysis had been performed there were 37 (8%) residual cavities. In noting the advantage of apicolysis in this series Blecher points out, however, that the character of the cases had changed during the years covered by the report. In the earlier years - when presumably apicolysis was not performed - there were more chronic cases operated on as a last resort.

Rogers, Shipman and Daniels (58) (1943) found that of 100 consecutive thoracoplasty cases, one third were left with a positive sputum. In some of these it was concluded that there was a residual cavity due to an underlying bronchial factor.

Alexander, Sommer, Trenton and Ehler (1) (1942) found 10(26%) cases with persistent positive sputum following thoracoplasty performed on 38 cases where tuberculous bronchial disease was present.

Opinions vary regarding the effectiveness of thoracoplasty as a means of closing persistent cavities when there is intermittent bronchial occlusion. Fre-:quently the operation has to be supplemented by such means as bronchoscopic suction or transthoracic needle aspiration or by the more advanced forms of cavernostomy.

Alexander et al (1) conclude that thoraco-:plasty is the safest and most effective treatment for the majority of cases where there is advanced bronchial and parenchymal tuberculosis. They are averse to

thoracoplasty where the disease is mainly in the lower portion of the lung and where much healthy tissue would be sacrificed. In all their cases thoracoplasty has been performed with bronchoscopic control, which includes bronchoscopic inspection in cases where tuberculous bronchitis is suspected. Although it would be expected that tension cavities should be difficult to close by means of thoracoplasty, in the experience of these authorities this has not proved to be the case when the cavity is situated in the upper third of the lung; in these cases complete cavity closure has been achieved. In spite of the presumed bronchial valve they have observed tension cavities deflate, sometimes incompletely however, during the stages of the thoracoplasty. Sometimes they have been obliged to perform surgical drainage in conjunction with thoracoplasty - on two occasions on account of bronchial stricture, out of 38 cases with tuberculous bronchitis; but apparently on a number of other occasions for a similar reason. The authors consider that pneumonectomy is preferable to thoracoplasty in certain cases where a tight undilatable stricture is accompanied by prolonged toxic symptoms from retained purulent secretions: thoracoplasty might increase the damming of secretions and produce a greater degree of toxicity.

Maier (41) (1945) on the whole favours thoracoplasty as a means of treatment of tension cavities. It is, in his opinion, preferable to artificial

pneumothorax. This author points out that sometimes the satisfactory closure of a cavity after thoraco-:plasty is only noted some months following operation. Whilst it is too early to evaluate the results of preliminary suction, on the whole Maier is averse to this practice, preferring to wait and see if the thoracoplasty will not be successful; even though he admits that secondary cavernostomy following thoraco-:plasty may be less effective than preliminary drainage (see p. 204).

Like Maier, Rubin (59) (1947) and Rafferty (55) (1944) consider that thoracoplasty is generally superior to artificial pneumothorax as treatment of tension cavities. Rafferty believes that it is the treatment of choice, with a needle at hand ready to deal with a pressure cavity should such arise during any stage of the operation.

Eloesser (25) (1940) and Vineberg and Kunstler (71) (1944) consider that thoracoplasty should not be performed for a tension cavity without previous decompression of the cavity. Vineberg and Kunstler have devised a very thorough technique whereby intra-cavitary pressures might be read before embarking upon a thoracoplasty. Should a tension cavity be discovered, aspiration is performed before carrying out the operation. Fuller reference will be made to this subject at a later stage.

Coryllos and Ornstein (20)(1939) referring to giant cavities, which are frequently caused by check

valves, expressed the belief that artificial pneumo-:thorax or thoracoplasty should be tried as it could never be estimated in advance what would be the result of the attempt: bronchial occlusion might be secured with the pneumothorax in which case the cavity would close with no further trouble. Thoracoplasty with wide apicolysis was considered to be the method of choice if pneumothorax failed: even then revision thoracoplasty had sometimes been necessary for a persistent cavity, having given good results in 29% of 43 cases. However, when the apex could be stripped, lowered and anchored with sutures or by introducing a muscle flap into the space formerly occupied by the apex, good results had been obtained in 35·3% of cases. As these results were not considered good enough, Coryllos tried other methods which will be described later.

The modern tendency to carry out fuller rib resection and wide apicolysis when performing thoracoplasty is proving advantageous. Hedblom and Van Hazel (29) believe that the need for a secondary antero-lateral stage for cavities persisting after thoracoplasty has been largely supplanted by the more modern thoracoplasty in which resection of the anterior portion of the upper ribs with their cartilages is combined with the posterior resection.

In 1933 Fischel (28) advocated the resection of all ribs above the lowest part of a cavity during

thoracoplasty in cases where there were active cavities with much secretion from their walls. Apicolysis with resection of the first rib had frequently resulted in compression of the bronchus without correspondingly compressing the cavity; the result was that secretions were dammed up in the cavity which was retained and enlarged. The modern thoracoplasty operation with its more extensive rib resection is less liable to leave room for this complication, which otherwise would be a suitable occasion for drainage of the cavity prior to thoracoplasty.

Pinner (53) (1945) concludes that the reasons for partial failure of thoracoplasty can be grouped under preoperative conditions and operative causes. Amongst the preoperative conditions which decrease the chances of sputum conversion and complete cavity closure are (1) cavities with a diameter of 6 cm. or more, especially if situated in the paravertebral gutter or in the lower lobe; (2) intrabronchial tuberculosis; and (3) pleural fibrosis when massive enough to prevent collapse. Operative causes are (1) insufficient collapse of the lung due to such causes as wound infection, which prolongs the interval between operative stages, or bronchial stenosis; and (2) failure to resect a sufficient number of ribs.

Brantigan, Aycock, Hoffman and Welch (12) devised a "relaxing thoracoplasty" operation which was

employed principally for the treatment of tension cavities, and cases with unilateral extensive disease from lung apex to base where the standard thoracoplasty was considered to be less suitable. The operation was unsuitable as a cure for giant cavities.

The authors believed that the bronchus was responsible for three conditions which might prevail within the lung at different times: (1) an increased intrapulmonary pressure, (2) a decreased intrapulmonary pressure and (3) atmospheric pressure conditions. In (1) there is a raised intrapulmonary pressure associated with spasm of the bronchial musculature or organic disease. If associated with the latter, a check valve mechanism has been formed (although it would appear that such a valve would be produced by both causes). In (2) there is a hypotensive state due to partial or complete occlusion of the bronchus as a result of organic disease of of decreased tonus of the bronchial musculature which permits a minimal air entry to the alveoli but allows the air to escape easily. A check valve exists which works in the reverse direction to the valve in (1). Obstruction may be complete resulting in air absorption. In (3) the bronchi may be free of organic disease or of spasm. Air passes freely in and out of the lung and atmospheric pressure conditions prevail. These three conditions are subject to frequent interchange.

Brantigan and coworkers conceived the idea of taking advantage of the contracting or hypotensive stage. If the restraining effect of the rigid thoracic cage were removed, when the hypotensive stage occurred the lung would be able to contract down unhampered. The same would hold true when scar tissue healing was in progress. The authors believed, moreover, that a relaxed thoracic cage was preferable to a rigid collapsed thoracic cage.

To produce the desired relaxation they sought to paralyse all the respiratory movements governed by the four dominants described by Monaldi: these are lines or directions of stress and motion related to the thoracic walls; they are defined as a superior vertical, an inferior vertical, and two lines of motion, one postero-anterior and another transverse-lateral. The desired effect was sought by removing the action of the muscles of the superior thoracic aperture, paralysing the diaphragm and carrying out anterior and posterior costectomies along the dominant line. This was carried out in 5 to 7 stages. The 7th stage was performed if there was disease from apex to base, when sections were removed posteriorly from ribs 8, 9 and 10.

The end results of treatment on 33 patients were as follows:

In 12 cases suitable for standard thoracoplasty, 10 had negative sputum. There were no deaths. In 21 cases unsuitable for standard thoracoplasty, 9 had negative sputum. There were 4 deaths (2 operative - of which one was with conversion to standard thoraco-:plasty - one from progression of disease and one from amyloidosis). The operation improved the cases clinically: many who had not been suitable for the standard thoracoplasty became so as a result of the operation.

In spite of precautions it was found to be very difficult to prevent bony regeneration, which largely defeated the purpose of the operation: in the series this happened to 13 cases, 8 of whom had a positive sputum at the time of the report.

The rationale of this operation is obscure, and the authors' reasoning with regard to the production of the hypotensive stage within the lung is difficult to follow. The operation was based largely upon the authors' belief that such a stage occurs. It will be agreed that a state of hypotension does occur under conditions tending to produce atelectasis: this will be generalised or localised according to the extent of the region involved. There is little evidence, however, to support their implication that a hypotensive check valve mechanism occurs with anything like the regularity which they infer.

Any advantage this operation might have over

the standard thoracoplasty, apart from its relatively minor operative stages, would appear to lie rather in that it abolishes the increased intrapulmonary pressure created by the first of the bronchial mechanisms which they describe: it removes the ability of the thoracic walls to create the negative extracavitary force which is the means of inflating the alveoli and a tension cavity. The success of the operation tended to be defeated in a number of cases, however, by the bony regeneration which is prone to occur.

The method might be regarded as having had a fair degree of success as a salvage operation: the operation does not commend itself, however, even in that category. It is a mutilating type of procedure, which must impose a good deal of tedium upon the patient who has to undergo the repeated stages required by the operation.

If it is not possible, or it is undesirable, to employ means such as have been described in an effort to alter the state of the bronchial check valve, or if such means have been tried and have failed, similar principles may be applied in spite of the valve: the cavity may be opened through the chest wall, or the cavity may be closed by blocking the bronchus at a site which may be remote from the valve.

II TREATMENT IN SPITE OF THE VALVE BY CREATING (a) AN ATMOSPHERIC OR NEGATIVE PRESSURE CAVITY THROUGH A TRANSTHORACIC APPROACH.

Writing in 1927 Lilienthal (39) referred to the fact that Sauerbruch, whilst operating, having accidentally perforated the pleura into a cavity made the best of it and found that the drainage thus afforded was of considerable benefit to the patient. In one of Lilienthal's own cases in which the draining bronchus became "angulated or plugged as a result of distortion" following a paravertebral thoracoplasty, the patient at once became toxic with a high temperature. Drainage was necessary. This was achieved by rib resection and opening the now putrid abscess, which formerly had been recognised as being typically tuberculous. Subsequent progress of the patient was good; healing of the fistula track proceeded so steadily that it became difficult to keep it open, and finally it closed. Four years later the cavity was apparently closed and the general health of the patient was fairly good. Lilienthal's conclusions at the time of writing were that opening a cavity through the chest wall was not the calamity that it had formerly been regarded: it should be avoided, however, if what he termed "drainage by compression" could be accomplished.

It was not until ten years later, however, that Monaldi and Eloesser, who may be regarded as the

pioneers, began to publish reports on their treatment of persistent cavities by transthoracic drainage. In 1938 Monaldi introduced his method of the closed suction drainage of cavities. Monaldi (43) had been impressed by the apparent readiness with which many cavities of considerable size were able to close with treatment. He concluded that the cause of this was two-fold: first, treatment had abolished the eccentric pull which previously had been imposed on the lung tissue which lay between the cavity and the chest wall; and second, a compensatory mechanism had been in operation on the part of the atelectatic lung tissue which had surrounded the cavity: this atelectatic tissue, by becoming aerated, had filled up the former cavity space. The atelectasis, to Monaldi's mind, was largely the result of there being created within a cavity on expiration, a positive pressure due to the narrowness of the bronchial outlet. In expiration the lung tissue surrounding the cavity became compressed between two converging forces - the positive intracavity pressure and the converging parietal force of the recoiling lung tissue (page 41). Monaldi conceived the idea that he could make use of the narrowing of the bronchus by instituting continuous suction within the cavity, thereby converting the positive pressure to a negative pressure. By this means he anticipated that he would be able to induce the atelectatic lung surrounding the cavity to re-expand and fill the cavity space.

Monaldi's original technique consisted of the introduction of a rubber tube into the cavity through a cannula and applying continuous hydrostatic suction.

As a result of researches which were in progress at that time (1939) Monaldi deduced that this method of treatment gave promise of other beneficial effects: conditions were created within the cavity which favoured rapid scar tissue healing. These were brought about through the improved circulation of blood in the area; the reconstitution of a normal $_{p}H$ of the secretions; and the elimination of the products of necrosis and with them the tubercle bacilli contained therein and in the cavity walls: all of which factors tend to ensure the coming together of vital tissues and healing of the cavity. Brunn (16) and other authorities have similarly noted the disappearance of tubercle bacilli from the cavities and drainage secretions when employing this method of treatment. Hudson (16), in the course of discussion, advocated washing out the debris from a cavity and dissolving the necrotic tissue by the use of enzymol, which is a digestive solution.

After a short follow up period the reports given by Monaldi at that time were encouraging: large cavities were induced to close; the patient became afebrile and gained weight. In 1942 Monaldi (44) was still claiming satisfactory results with this method of

treatment; though statistical support for these claims was not produced. He admitted that at the end of treatment the draining bronchus generally remained open and advocated introducing a sclerosing agent into the cavity at the end of treatment in order to obtain permanent closure of the draining bronchus. Results following this procedure he considered to be satisfactory.

The extent of Monaldi's claims, based on his personal experience, were that immediate beneficial results are obtained, but permanent cures may be obtained also if the treatment is correctly applied. Broadly, the indications given for closed suction drainage were: (1) isolated cavities surrounded by healthy lung tissue; (2) stationary cavities with inactive disease in the surrounding tissues; (3) lesions with multiple cavities provided they fit into one of the other suitable categories. The method is contra-:indicated in "biological cavities" in which there is much loss of tissue and the pericavitary tissue is diseased: also in the presence of fibrothorax. The method is indicated in cavities in the formation of which mechanical factors are prevalent with resulting atelectasis.

Kupka and Bennett (37) (1940) reported on the treatment of 17 cases by Monaldi suction aspiration. As 10 were still receiving treatment the results at that time were inconclusive. Permanent closure of

the cavity, however, was only recorded in one case after six months observation. Reduction in the size of the cavity, symptomatic relief and improvement in the patient's general condition were observed: the operation was beneficial as a preparation for thoraco-:plasty.

Brunn, Shipman, Goldman and Ackerman (16) (1941) employed this method - which they termed "trans-:pleural decompression", employing continuous suction through a catheter attached to a mechanical pump - on 20 cavities in 18 patients. As with Kupka, treatment was inconclusive, but only 3 cavities were apparently closed at the time of their report. Several of the cavities were rid of their tuberculous infection however.

Rubin (59) (1947) believes that it is seldom a cavity closes completely with drainage alone. At its best, such a method may be used as a preparation for thoracoplasty, but even for that purpose it has its limitations on account of the fact that most cavities are situated posteriorly and posterior drainage would interfere with the subsequent thoracoplasty operation. Rubin concludes that "on the whole the Monaldi operation has little to offer" and that "in borderline cases it is preferable to enlarge the indications for thoracoplasty".

Rafferty (55) considers that after a cavity has been treated by closed drainage it should be opened

widely and packed to cause complete and permanent occlusion of the broncho-cavitary junction and allow the cavity to heal by granulation.

Many authorities have achieved encouraging results employing Monaldi's method of treatment. Most authorities are agreed, however, that although cavity closure may be secured by this means, it usually opens again when the treatment is discontinued. In view of the undoubted temporary success of Monaldi drainage the procedure is often adopted as a prelude to thoracoplasty in cases of giant cavities or of large tension cavities: in both cases the pericavitary tissues must be capable of re-expansion, mainly by means of reaeration of atelectatic air cells. Having secured temporary closure, the major surgical collapse of the lung is carried out in order to maintain the advantage thus gained; at the same time the danger of producing a thoracoplasty collapse around a tension cavity, is obviated.

Vineberg and Kunstler (71) (1944) had had 12 residual cavities in 119 thoracoplasties (1942) and in these cases it was found subsequently that the intra-:cavitary pressures were highly positive. The authors considered that intracavitary pressures should be recorded in all cavities larger than 2.5 cm. diameter before embarking upon a thoracoplasty. Should the results indicate a positive intracavitary pressure, a thoracoplasty should not be performed until a prior

course of transthoracic suction drainage had been carried out in order to close the cavity. For this purpose Vineberg and Kunstler devised a most careful technique for cavity puncture, which was controlled by saline suction in case of the needle entering a blood vessel, and so regulated that no air was lost from the cavity during the process of recording the intracavitary pressures, as an error of this nature would vitiate the results. Following the preliminary needling, and having made certain of pleural symphysis, a catheter was intro-:duced into the cavity to which it had been decided to apply suction, and continuous suction was carried out for twelve hours a day, or sometimes all day and night, for a minimum period of ten days. This was followed by a period of withdrawal of the catheter, which covered three to four weeks. The authors expressed the view that in cases where there were large apical cavities which were firmly adherent to the anterior chest wall, a preliminary anterior stage thoracoplasty should be carried out before drainage, as such cavities would be prevented from collapsing whilst being held open by adherence to the chest wall. The authors treated 27 cases of proved positive pressure cavities in this way without a single complication of haemorrhage, empyema, spontaneous pneumothorax or air embolism. Of 54 cases investigated, 24 had had tension cavities treated by

means of drainage followed by thoracoplasty, with 67% closure of cavities; whilst in 30 non-tension cavity cases which had been treated by means of thoracoplasty alone cavity closure had occurred in 91%.

Vineberg and Kunstler argued on the principle that it was no use collapsing a lung in an attempt to close a cavity by releasing extracavitary forces when that cavity was being ballooned out by a high internal pressure which was the result of a bronchial check valve mechanism. The problem should be approached from the point of view of undoing the raised intra-:cavitary pressure. Collapse procedures in such cases, they maintained, merely altered the location of the cavity,without influencing the raised intracavitary pressure. Whilst admitting that "a cavity having a positive pressure before thoracoplasty may become a nega-:tive pressure cavity after the first or second stage," they added, "It is difficult to see how a diseased bronchus would be very much changed by dropping the cavity down to the level of the 3rd or 4th dorsal vertebrae". It is pointed out, however, concerning this argument, that under such circumstances a cavity is not likely to be dropped without the mechanics of the draining bronchus being affected by the shortening and possible collapse of that bronchus: this might well take the form of complete occlusion of the bronchus with subsequent closure of the cavity. Admittedly there is no way of

knowing whether the cavity will close or merely balloon out more under these circumstances. Whilst there is much that is commendable about the precautions of Vineberg and Kuntsler, hesitation must be felt re-:garding embarking upon diagnostic intracavitary needling in every cavity larger than 2.5 cm. before undertaking thoracoplasty. A more practicable procedure might be to follow the course adopted by these authors before per-:forming thoracoplasty in those cases where clinically and radiologically there are indications suggestive of a tension cavity.

One method of treating persistent cavities, which will be mentioned only to be condemned, is that of induced cavity rupture by needle aspiration through the chest wall across a pneumothorax space, as recomm-:ended in certain instances by Erwin (27) (1944). The author apparently considered closure of the cavity at the expense of the inevitable pleural effusion, which, however, did not generally become a troublesome empyema, a profitable exchange. A warning is issued against using this procedure if the draining bronchus is patent because of the danger of a broncho-pleural fistula forming; yet one of the indications given for this treatment is in cases where the bronchial disease favours cavity distension.

If the tension cavity persists, or shows its true nature for the first time, after thoracoplasty, consideration will have to be given to drainage after the operation. Maier (41) (see p.189) considers that

a good deal of difficulty is liable to be encountered in securing cavity closure and complete wound healing following secondary cavernostomy after thoracoplasty. The pericavitary lung tissue might respond poorly to reinflation for the purpose of filling in the cavity space, he maintains, especially if the thoracoplasty has been performed for some considerable time. Maier criticizes suction drainage prior to thoracoplasty on the grounds that it does not affect the bronchus: there is nothing to prevent the bronchus from opening and reinflating the cavity later on.

At about the same time as Monaldi was formula-:ting his method for the closed suction drainage of cavities, Eloesser (24) (1938) was experimenting with similar methods: these included the open drainage and packing of cavities which had failed to close by the usual means. Eloesser realised that a one-way valve mechanism was frequently the cause of these persistent cavities. Unlike Coryllos, he believed that to convert the partial obstruction into a complete one was to court trouble. On the other hand he wrote "Opening the cavity to the outer world by means of a trochar or catheter may open a previously occluded bronchus or may release a stop valve mechanism". As a result of his investigations of persistent cavities, in which the underlying cause was of bronchial origin, Eloesser concluded, "Closed suction drainage of blocked cavities, a method which at the outset seemed most promising and

reasonable, has been a failure". Eloesser further concluded that open packing of such cavities, which included opening and packing the cavity or packing it from outside the cavity wall, was curative but restricted in application and troublesome. Thoracoplasty was included in the treatment of six of the nine cases he described. Eloesser doubted the efficacy of this operation in such cases: apicolysis he considered inadvisable because it merely caused displacement of the cavity. This authority uttered prophetic hopes that resection, which he looked upon as the ideal method of treatment, might become a safer operation in the future.

Eloesser, as has been said, believed that a cavity could be patent and yet the bronchus remain closed. He expressed this view with reference to the first case he described in a series of persistent cavities which he had treated by various means (24). In this particular case a cavity had refused to collapse in spite of an artificial pneumothorax and a thoracoplasty having been tried. Lipiodol and methylene blue which had been injected into the cavity, were not expectorated. Eloesser concluded that it was unlikely that the cavity communicated with a bronchus at all: the cavity resisted collapse measures and did not heal. Belief in this principle possibly accounted for the disappointment felt regarding closed suction drainage of cavities. As Coryllos pointed out (20), referring to this particular report given by Eloesser, suction merely opened the bronchial valve whereupon air

was drawn continuously through the bronchial tree and through the cavity. Eloesser (25) subsequently admitted this fact, though it is evident it was recognised at the time of the experiments.

But Eloesser did not abandon suction drainage nor thoracoplasty as methods of treating persistent cavities. By 1941, he was advocating the combined use of these two - to his mind - dubious forms of treatment (25). Rogers was recommending preliminary aspiration of a tension cavity in order to secure closure, followed immediately by thoracoplasty to maintain the advantage gained by the suction. This method was favoured by Eloesser. At the same time, the latter was using a "skin flap" operation, devised as a means of keeping the fistula track open when it was desired merely to keep the cavity open to the atmospheric pressure. A U-shaped skin flap was attached to the border of the cavity after 5 cm. of overlying rib had been resected. Two such cases are described. Suction drainage having failed, the patients were sent home with a sinus from which came a T.B. negative discharge. Except in the case of extrapleural pneumothorax, Eloesser was at that time averse to compression methods of cavity closure, such as gauze packs, because of the danger of perforating the cavity.

It was not long before other authorities were publishing their results of treatment by means of

the "Eloesser skin flap" operation. In 1941 Brunn and coworkers (16) reported on a case in which there had been a cavity in the right upper lobe. Artificial pneumothorax had failed. Following a two-stage thoracoplasty the cavity had become bigger than ever; intracavitary pressures recorded were plus 20 plus 12. A needle was inserted and the cavity deflated; but every time the needle was withdrawn the cavity ballooned up again. It was decided to make a permanent opening to the atmosphere. Accordingly a flap drainage operation was carried out after the manner described by Eloesser, and the cavity was packed with iodoform gauze. Six months later the sinus had closed: nine months from the operation the cavity closed and the flap healed.

In 1943, Rogers, Shipman and Daniels (58) reported on nine cases in which cavities had persisted after a second-stage thoracoplasty. Concluding that the bronchial factor was responsible, flap drainage and packing of the cavity as described above, were performed. At the time of their report, five of these cases had been operated on more than six months previously; the results were that all five had sputum conversion and the wound closed. The other four cases were making promising progress. By employing such a procedure, the authors concluded that they were able to sacrifice the minimum of healthy lung and avoid "mutilating and hazardous" procedures. Monaldi suction drainage had not proved a success in their experience.

Encouraged by the report of Rogers and coworkers, Randolph (56) resorted to flap drainage in two cases which he considered unsuitable for other forms of further treatment. The first case had had bilateral extrapleural pneumothorax performed for large cavities. This treatment had failed on the left side where a large cavity was to be seen in the region of the junction of the middle and upper zones. Following flap drainage the lung re-expanded, the cavity disappeared and the sputum was T.B. negative. The patient was well four years after operation. It is to be noted that the patient had been kept fairly strictly in bed for two years following operation. In the second case there was some questionably active disease in the right lung, but no cavitation mentioned. In the left lung there was a large cavity at the level of the 4th - 6th ribs posteriorly. Artificial pneumothorax had been abandoned. Extrapleural pneumothorax was embarked upon but, the movement of the lung at operation being considered excessive, a skin flap operation was performed in two stages. The cavity subsequently closed fairly rapidly but the patient had rather a long and stormy postoperative period. From the report given, it is to be wondered whether a left upper thoracoplasty might not normally have been undertaken in this case.

It is questionable, however, whether the ultimate results would have been any better, if as good, had this been done. Randolph considers that flap drainage should be reserved for cases which are unsuitable for other methods of treatment - such as certain cases with tension cavities. It is a simple procedure and to be preferred to Monaldi drainage. Cavity drainage, according to Randolph, is indicated in cases where there is cavitation in the lower part of the upper lobe, a middle lobe or upper part of a lower lobe: in such cases too much healthy lung would be sacrificed during collapse measures. This authority considers that success with open drainage of cavities is unusual and ordinarily it has been disasterous.

By 1945 some of the earlier advocates of flap drainage were expressing dissatisfaction with this method of treatment. Eloesser, Rogers and Shipman (26) were to be found confessing that incision and drainage of cavities had proved to be unsuccessful. They believed, in fact, that when opened widely there was a tendency for a cavity to be drawn into a larger hole by the retractive pull of the surrounding tissues: drainage or deflation of cavities, if necessary, should be carried out through a small opening, at the same time applying some device which would keep the intracavitary pressure negative. Closed suction they considered to be good, but it required constant supervision, and the suction must overcome the

leak of air into the cavity from the bronchus. Accordingly the authors devised a valvular modification to the flap method of cavity drainage. This operation, which is carried out in two stages is so devised that the tip of the flap which formerly was anchored to the cavity wall, is now free and is so placed in the cavernostomy opening that it permits air to pass out of the cavity if the internal pressure rises but closes the opening by the force of the atmospheric pressure when the intracavitary pressure is negative. The flap is left until the cavity has closed. After a year or more, the flap is detached and sewn back into its original position. The authors were encouraged by their results. The method was employed after thoracoplasty in upper lobe cavities and after phrenic paralysis in lower lobe cavities. They claimed 18 successful results out of 23 cases treated. There were two deaths as a result of meningitis.

In spite of the opinion of Eloesser, Rogers and Shipman, open cavity drainage continues to be tried. More recently, in 1947, O'Brien, O'Rourke, Test and Skinner (46) published the results of their treatment of 74 cases by means of open surgical drainage of cavities. The operation was looked upon as a "Salvage" operation in cases where other means had failed or were contraindicated for reasons such as inadequate cardiorespiratory reserve. These authors

preferred not to use the skin flap method of Eleosser as they considered it was preferable to avoid, if possible, a permanent fistula. The indications for treatment, in the 74 cases, were placed into four groups as follows:-

I. Inadequate cardiorespiratory reserve - 43 patients

II. Residual cavitation following thoracoplasty - 13 patients

III. Lower lobe cavity - 8 patients

IV. Other conditions - 10 patients.

Group IV included 5 cases converted to open drainage where the Monaldi operation had failed. Of the total 74 cases treated the operation was incomplete in 6 owing to such interfering complications as pleural effusion: this left a total of 68 completed cases. All 68 cases had advanced tuberculous disease: 36 were looked upon as poor surgical risks; 15 as fair, and 17 as good surgical risks. In view of these figures it is not surprising to find only 32 cases still alive over one year after operation. The operation consisted of limited rib resection followed either by packing the cavity or by inserting a firm rubber tube into the cavity until the bronchial fistula closed. The best results were obtained in Groups II and III, in which most of the patients were considered to be fairly good surgical risks. In Group II the original 13 cases were reduced to 8, as 3 died and 2 had incomplete operations: of these 8, 6 were arrested, one improved and

in one other it was too early for classification. The authors regarded a case as being arrested when the wound had healed without drainage, the cavity had closed on X-ray series and sputum had remained T.B. negative for a minimum of 8 months. In Group III the 8 cases were reduced to 6 owing to failure of the operation to achieve cavity drainage in 2 cases: of these 6, 5 were arrested and one had improved. Of the entire series, taking 32 survivors of over one year, 56% had healed, 19% were healing, and 25% remained unhealed. The authors concluded that cavernostomy was useful in patients of Group I provided they were not dyspnoeic. As has been stated, the other more orthodox means of treatment had been tried in these cases. Cavernostomy had been used as a final resort. Furthermore, the patients, taken as a whole, were in poor general condition. Agreement will be felt with the authors, that the operation is worthy of trial in such difficult cases as they describe, especially those cases in Groups II and III. It is to be noted, incidentally, that only one of the cavities in the whole series is described as being a tension cavity. The impression gained from a study of the report is that several of the cavities in all four groups of these cases might come under this category.

II. TREATMENT IN SPITE OF THE VALVE BY CREATING (b) A CLOSED BRONCHUS THROUGH OCCLUSION AT SOME SITE OTHER THAN THE VALVE.

Many attempts have been made to close cavities by direct bronchial occlusion, either by means of some sclerosing agent or by the introduction of some foreign body which will act as an effective obstruction.

Eloesser (25) (1940) reports that Rogers tried injecting 0·6 cc. of 10 - 20% silver nitrate into cavities in two cases following aspiration and thoracoplasty, in an attempt to seal the bronchus, but failed.

Coryllos and Ornstein (20) (1939) because of the poor results obtained in treating giant cavities, tried to close the bronchus by two different approaches: one was by the injection of substances into the cavity through the chest wall, or cauterizing through a cavernoscope the bronchial opening into the cavity; the other approach was opening the cavity widely during revision thoracoplasty and filling the space with a large pedunculated flap taken from the paravertebral muscles. In six cases, a needle was inserted into the cavity and one of the following substances was injected: copper morrhuate, blood - either plain or mixed with thrombin, 30 - 60% silver nitrate, 8% gelatine, and 1·5% agar. It is not clear from the report whether one substance was injected into each

cavity or more than one was tried in one cavity. In no case was a satisfactory result obtained. In three other cases, the following methods were tried through a cavernoscope: in I, 60% silver nitrate was applied to the bronchial orifice to the cavity; in II, the mucosa of the orifice was treated by means of galvanocaut-:ery; in III, Monolate - a sclerosing substance used for the treatment of hernia - was injected around the orifice of the draining bronchus. None of these methods was found to be effective. In applying the second method of approach to the problem, intracavitary transplantation of a muscle flap was tried in twelve cases. Although the procedure was found to be effective, producing a negative sputum in five of the cases, it was liable to dangerous complications.

Thomas, Gough and Still (69) (1943) published a preliminary report on the treatment of six cases in which Monaldi drainage had failed, by introducing plasma into the cavity and the draining bronchi through the drainage tube. The plasma was allowed to clot thus promoting occlusion of the draining bronchi. Complete cavity closure was obtained in two cases; but only a short period of observation had elapsed at the time of the report. Although the authors concluded that the results had been promising and had been "found in some measure to be successful" it was felt to be too early at that time to form any conclusion.

Brooks (15) (1938) produced broncial occlusion by means of a small rubber balloon attached at the end

of a ureteric catheter. This was introduced into the bronchus through a bronchoscope and, when in position, inflated with 4 cc. distilled water. The preliminary report is given of this method having been used in a case treated by right artificial pneumothorax. A cavity had been present near the apex of the lower lobe. One week after the induction of the pneumothorax, the balloon was inserted into the lower lobe bronchus in an endeavour to produce a selective collapse on the cavity area. The balloon was left in situ for six hours, then withdrawn. The pneumothorax was continued as usual. At the end of six hours selective collapse of the lower lobe and cavity had occurred and remained during the two months follow up period.

Monaldi (44) injects a mixture of charcoal and cheratin (5:1) dissolved in collodion into the residual cavity at the end of intracavitary aspiration in order to obtain permanent closure of the draining bronchus. The results are considered to be satisfactory but no statistics are quoted to support these claims.

PULMONARY RESECTION.

Writing eleven years ago upon the subject of "Blocked cavities in pulmonary tuberculous", Eloesser expressed the view that "excision" was the ideal method of treatment only it was, as yet, too risky: it might become safer later on.

Pulmonary resection, as a method of treating persistent cavities, is a confession of the failure of the methods of cavity closure to provide a reliable course of action when faced with this problem. Since by resection the cavity is removed bodily from being able to continue to defy attempts at closure, the success of this method is the success and safety of the operation as a surgical procedure.

Pulmonary resection to-day is in its infancy as a surgical procedure. It is evident, however, that it is steadily becoming a safer operation and that it is establishing for itself a secure place amongst the methods of thoracic surgery.

Overholt and coworkers (48) writing in 1947 expressed the view that the rapid growth of pulmonary resection in tuberculosis had largely been due to the fact that it had been needed. The relative ineffectiveness of the various collapse procedures in the control of many tuberculous lesions, they maintained, was now generally recognised: this was especially true in relation to certain conditions, amongst which were included endobronchial tuberculosis and tension cavities. The need also arose in respect of the large number of thoracoplasty failures still requiring treatment.

In the opinion of Bailey (6) (1947) collapse therapy is contraindicated, and actually dangerous, when there is stenosis of a large bronchus with definite retention of secretions, or a persistent check valve or

retention cavity: in such, resection is often the method of choice.

In 1945 Overholt and Wilson (47) included the following amongst the occasions when they considered pulmonary resection was the treatment of choice: (1) in preference to thoracoplasty in cases of active parenchy-:mal disease complicated by bronchial stenosis; (2) in preference to cavernostomy in the treatment of tension cavities in unilobar or extensive unilateral disease; (3) unilateral disease that has failed to respond to thoracoplasty; (4) extensive basal tuberculosis. Tuberculous bronchitis, active or inactive, was an indication rather than a contraindication for resection, Writing two years later, Overholt (48) continues to include these indications, adding, with reference to basal tuber-culosis, that resection was an indication when other simpler measures had failed or were contraindicated.

As yet the results of treating persistent cavities by resection must, for the most part, be gauged from the results of treatment by this method in general. Sometimes more direct reference is made to the results when treating tension cavities. Maier (41) (1945) reports having performed sixteen lobectomies, eight of which were for tension cavities: ten had an uncomplicated postoperative course and there was one fatality. Maier had performed two pneumonectomies for tension cavities; one where revision thoracoplasty had failed and one where artificial pneumothorax had failed: in both cases there

had been an uneventful postoperative course.

Bailey (6) (1947) reported on the results of pulmonary resection in 80 cases: 41 pneumonectomies with 15 (36%) deaths; 32 lobectomies with 8 (25%) deaths; 7 segmental resections with no deaths. The total mortality rate for the whole series was 27%. These figures covered a four year period and not all the deaths were due to operation nor even to tuberculosis. Bailey pointed out that these figures did not give an accurate picture of the mortality and morbidity since the earlier cases had been dealt with by "antiquated and de-:ficient methods". Included in this series were 9 cases with serious bronchial stenosis which had been treated by pneumonectomy with two deaths; 12 "very large" cavities treated by pneumonectomy (5 cases with one death), lobectomy (6 cases with two deaths) and segmental resection (one case with no death); 3 "check valve" cavities treated by lobectomy (2 cases) and segmental resection (one case), with no deaths. No description is given of the "very large" cavities to know how or why they were distinguished from "check valve" cavities.

Overholt and Wilson (47) (1945) reported on 60 cases of pulmonary resection; 36 pneumonectomies and 24 lobectomies, with a total operative mortality of

11·6% (4·3% in 47 "reasonable risk" cases and 38·5% in 13 "desperate risk" cases). The authors regarded the operation very favourably in the light of the more up-to-date technique in which hilar structures are ligated individually and the stump reinforced with a flap of pleura, whilst use of the stump tourniquet has been abandoned.

More recently (1947) these authors and their coworkers (48) have reported on resection treatment of 88 patients (92 resections) between 1934 and 1944. There have been 58 pneumonectomies and 34 lobectomies; 68 (74%) in reasonable risk cases and 24 (26%) in desperate risk cases. This series presumably includes the cases in the 1945 report. The follow up period ranges from 2 - 12 years. It is interesting to note that 74 of the resections have been performed since 1942, whereas only 18 had been performed prior to that date. The authors point out that the duration of time since operation is too short for the operation to be considered as a late follow up: only when a large number of patients have been followed for over five years will the late statistics be really significant. Reference has already been made to some of the indications for resection according to these authors. In this 1947 series, failed thoracoplasty was an indication for resection in 16 (17%) cases, in 5 of which tuberculous bronchitis was present. Of the whole series

35 (38%) cases had a complicating tuberculous bronchitis. The commonest indication was extensive predominantly unilateral tuberculosis which comprised 42 (46%) of the cases. Of these 42 cases, 26 (62%) had tuberculous bronchitis. This group also included large tension cavities, multiple cavities throughout the lung and a combination of apical with basal cavitation.

When the operative mortality recorded in 1945 is compared with the corresponding figure given in 1947, there is little significant change to be noted. In 1945 the total operative mortality was given as 4·3% in reasonable risk cases and 38·5% in desperate risk cases: in 1947 the corresponding figures are 5% for reasonable risk cases and 43·8% in desperate risk cases. Yet when the figures quoted are examined more closely and compared with earlier results still, there are indications of a fall in the operative mortality since 1934: this has been more striking with reference to the pneumonectomy cases when consideration is given to the most recent figures for 1944-46. Overholt regards 60 days as the postoperative period. Of the 34 lobectomy cases of 1947, 3 (9%) died during the postoperative period: the operative (or postoperative) mortality rate fell from 29% prior to 1942, to 4% during the following two years to 1944. Percentages however, lose their significance when referring to only three cases. Of the 58 pneumonectomy cases, 13 (22%) died postoperatively:

the operative mortality fell from 27% prior to 1942 to 21% during the next two years. Overholt also included a summary of the most recent results during the 2 years 1944-46. Lobectomy was performed in 35 cases during these 2 years with 2 (6%) postoperative deaths: pneumonectomy was carried out in 69 cases with 9 postoperative deaths, thus reducing the operative mortality rate to 13% for this operation (4% for reasonable risk cases and 44% in the desperate risk group). Reviewing broadly the results on the 88 patients whose histories have been followed for 2 - 12 years, 42% of the lobectomy cases are clinically well with a negative sputum, whilst 18% are dead: 48% of the pneumonectomy cases are clinically well with a negative sputum and 41% are dead. The authors conclude that the results following pneumonectomy have been more gratifying than those following lobectomy. The disappointing results following lobectomy they attribute largely to the high incidence of ipsolateral exacerbation, which in turn is attributed to poor judgment in applying the procedure rather than to the operation itself. The true value of these operations can only be realised in the light of the presence of the desperate risk cases included in the numbers treated. In the words of the authors, "We feel that any salvage in this group of patients, regardless of how small, can be considered pure gain".

TREATMENT DIRECTED THROUGH THE NEURO-MUSCULAR SYSTEM OF THE LUNG.

Although the basis of the forms of treatment included under this heading, is at present hypothetical, nevertheless some astonishing results have been obtained, mainly by workers on the Continent of Europe, by means of treatment which so far can only be explained upon a neuro-muscular basis.

At a meeting of the Societé D'Études Scientifiques sur la Tuberculose, of which a report was published in 1946, Braillon (11) described the results of treating 26 cases of cavities persisting in the presence of artificial pneumothorax, by means of faradism applied to the lung. The faradic current was applied by means of a small gold chain introduced through the chest wall by means of a cannula and allowed to rest lightly on the pleura in the region of the cavity. The current was applied 4 to 6 times each session at different points near the cavity. Treatment was generally repeated 4 to 8 times during the course of 7 to 15 days. The cavity was observed to shrink progressively and disappear in 15 days to 4 months; taking on an average about 6 weeks to close. The best results had been obtained in cases where there were no pleural adhesions. There were pleural adhesions present in 7 of the cases treated; in 19

cases the lung was free. In the first series with adhesions, 2 cavities disappeared; whilst two others only closed following a repeat of adhesion section. In the second series with no adhesions present, 5 had been treated sooner than was usual after adhesion section, with success; 15 had been treated 2 to 7 months after adhesion section with cavity closure in 13. On 10 occasions in 6 cases faradism was followed by what was described as atelectasis of the whole lung which commenced about 12 hours after treatment and lasted 4 to 6 days. The lung on these occasions appeared completely dark when viewed radiologically, was retracted and immobile: there was no pyrexia, no dyspnoea and the intrapleural pressure -2—zero. It is a curious fact that the intrapleural pressure did not become more negative if the lung was atelectatic and retracted; and yet it is difficult to find any other explanation for the phenomenon as it is described in the report. Braillon could not find a satisfactory explanation for the response of the lung to faradism. He considered that apparently pleural irritation from a faradic current acted upon the nervous control of the bronchial tree. It was not thought that the mechanism consisted of bronchial spasm leading to intracavitary atelectasis because the cavity closed slowly: also a fluid level was never seen after treatment; on the contrary, a fluid level, if it had been present, disappeared, indicating an open bronchus.

In the discussion which followed this report, de Léobardy recalled Kindberg's method of treating cavities which persisted in spite of a good artificial pneumothorax: this was by means of small quantities of gold salts instilled into the pleural cavity. By this means enormous cavities had been induced to close. In some most unlikely cases with a defective collapse, chemical irritation of the pleura in this manner had been seen to produce thickening of the cavity walls which finally obliterated the lumen; this fact subsequently having been confirmed by tomography.

According to Vallentin (70) such phenomena are explained on a basis of a pleuro-pulmonary reflex whereby the pulmonary muscles, especially those which he believes surround the alveoli, can be induced to contract by means of chemical or mechanical stimuli, including the products of bacterial metabolism. There is considerable doubt, however, regarding the presence of any muscle fibres beyond the alveolar ducts (p. 2.): this need not necessarily entirely discountenance Vallentin's hypothesis and would still provide an adequate explanation for the atelectasis observed by Braillon. Vallentin describes the French clinicians as having recommended for some time the use of gomenol in cases of incomplete pulmonary

collapse. As a result of the improved collapse following the instillation of this irritant substance into the pleural space, persistent cavities had sometimes been induced to close completely.

Bariety, Lesobre and Choubrac (7) (1943) described a case where there was a tension cavity apparently in or near the upper zone, which ballooned up alarmingly following artificial pneumothorax and adhesion section. This condition was treated by a combination of frequent, small refills and intravenous atropine. Daily injections of 0·5 mgm. atropine suplhate were given for 15 days. Four days after commencing the injections the cavity was smaller, whilst the base of the lung was re-expanding. From this time onwards the upper lobe collapsed progressively and with it the cavity. The authors were unable to find any adequate explanation for this phenomenon. Atropine, they pointed out, acts by diminishing broncho-pulmonary tone, favouring relaxation of the bronchial muscles and expansion, not retraction, of the lung. They speculated upon the possibility of the atropine having suppressed the mechanical causes within the bronchus of the ballooned cavity, whilst the repeated refills brought about collapse of the affected lobe: the atropine appeared to them to have played a part in the selective collapse of the diseased lobe. Reports on a number of cases would be needed

before formulating any conclusions on this method of treating tension cavities. Examining the report given on this case the impression is that the cavity closed and the upper lobe became atelectatic as the result of complete occlusion of one of the larger bronchi: atropine may have had nothing to do with the process at all, beyond perhaps drying up the secretions within the bronchus. It is not necessary to maintain that lobar atelectasis must always occur suddenly. The repeated refills in conjunction with dried intrabronchial secretions might have been responsible for converting a check valve into a complete block of the airway. On the other hand, a just criticism cannot be made in this case from reading a report; the authors watched the proceedings from day to day. There are however enough features in the report alone to call for further investigation into the effect of atropine upon such cases.

C A S E S

CASES.

Case I.

Name G.P. Age 17. Occupation. Draughtsman.

Admitted. 6th February 1946. Discharged.23rd November 1947.

Past History. Measles, whooping cough, jaundice.

History on admission. Cough commenced 2 months before admission followed 2 weeks later by haemoptysis. Patient was in bed 3 weeks at home, then allowed up. No cough now, 4 to 6 pieces of sputum daily, good appetite and states he has gained weight.

Family history. Father and maternal grandfather have had rheumatic fever - with cardiac affection in case of father.

Progress Notes.

On admission the sputum was T.B. negative. The patient was mainly afebrile throughout the course of his stay in the Sanatorium, but mouth temperature occasionally rose to 99°F. during the first 2 months.

Preadmission X-ray (2.1.46) Showed disease confined to the right lung where there was infiltration and consolidation mainly in the root area of the mid zone, with cavitation above and below in this area. The cavities were irregular in outline (moth-eaten) and there was evidence of more tissue reaction around the lower cavity or cavities which appeared to be mainly areas of excavation in consolidated lung.

X-ray on admission (8.2.46) (Plate I). Scoliosis with convexity to the left. Heart. Slightly pronounced pulmonary infundibular shadow. Left lung. Normal. Right lung. Increased consolidation around the cavities which conformed to type I of Ornstein, and of Pinner (p. 68, 133)

(11.2.46.) Right artificial pneumothorax induced.

(3.4.46))
) Sputum T.B. negative.
(2.5.46.))

(4.6.46.) Sputum positive (Gaffky I) for the first time.

(21.6.46.) X-ray showed a good collapse with no visible adhesions.

(8.8.46) Sputum T.B. negative.

(31.10.46) Sputum again T.B. positive (Gaffky IV)

(25.11.46) Thoracoscopy. No adhesions seen.

(5.12.46). Sputum T.B. negative.

(6.12.46.) X-ray shows questionable cavitation right root area.

(13.12.46) X-ray taken with increased penetration (Plate II). Cavity to be seen in lower part of right mid zone. Cavity smooth and spherical in outline, size of a cherry, surrounded by a definite wall probably composed of reactive and atelectatic lung tissue. The outline of the draining bronchus can also be

made out. There is considerable clearing of the reactive area about the root zone.

(Same date) Left oblique X-ray shows cavity near mediastinum, consequently lying posteriorly. Cavity wall irregularly spherical. The appearances about the triangular heart shadow in the right cardio-phrenic angle are perhaps suggestive of adhesions to diaphragm in this area.

(14.1.47) Sputum T.B. positive (Concentration test.)

(3.3.47 and thereafter) Sputum T.B. negative.

(10.3.47) Bronchoscopy. "Right dorsal lobe orifice full of pus. After this had been sucked away, the orifice was seen to be very red and inflamed". The bronchoscopist expressed the opinion that this was a bronchial lesion.

(13.3.47) Right phrenic crush performed.

(11.4.47) X-ray shows right diaphragm raised 1¼". Questionable cavitation.

(23.4.47) Pneumoperitoneum induction. With the pneumoperitoneum, the right diaphragm rose a further 2", making a total rise of 3¼". Thereafter (Plate III) no cavitation seen on X-ray; but one film gave a doubtful impression.

(23.11.47) Discharged home. Whilst in the Sanatorium, although a pale youth, the patient's general condition had been fairly good. He had been ambulant much of the time and had had no significant symptoms.

In April 1948 he was considered fit to do part-time work in a drawing office.

(27.5.48) X-ray shows considerable re-expansion of the lung and drop in the diaphragm. The appearance of cavitation is still uncertain.

(19.7.48). Tuberculosis Officer writes that the patient is well and sputum was T.B. negative (Concentration) one month previously.

SUMMARY.

In this case a cavity of the "moth-eaten" variety and situated in the lower lobe failed to close in spite of what appeared to be a perfectly satisfactory artificial pneumothorax. Suspicions were aroused by the presence of an occasional positive sputum and not until then was a closer scrutiny made for residual cavitation. Definite cavitation was seen on the X-ray of 13.12.46, the cavity now having a spherical appearance. Apart from this time, since collapse therapy had started any suggestion of cavitation to be seen radiologically was very uncertain. Bronchoscopy showed mucosal inflammation with purulent discharge in the dorsal lobe bronchus, which further confirmed the likelihood of a cavity persisting in the region described. Following elevation of the right diaphragm and continuing the pneumothorax, the indications are that the cavity is now closed.

DISCUSSION.

Without a lateral X-ray film it cannot be known how much of the disease was centred around the hilar structures and how much lay in a position anterior or posterior to the root level. In addition, a lateral film might have shown whether or not the root glands were enlarged. From the films available, however, it seems reasonable to presume that the root area was involved in the tuberculous process, probably to a considerable, if not maximum, extent: that having been the case, one or more of the major bronchi were liable to have been involved. In addition, enlarged hilar glands might have been present, especially in view of the age of the patient. These factors contraindicated an artificial pneumothorax. As it turned out, however, no massive pulmonary collapse from occlusion of a major bronchus, occurred. Phrenic paralysis, probably supplemented by pneumoperitoneum, was the correct initial treatment in this case.

Definite cavitation was seen on X-ray (13.12.46) otherwise the possibility of tuberculous endobronchitis might have been considered the cause of the occasional positive sputum.

The persistent cavity assumed the appearance of either a positive pressure cavity or a cavity with walls composed of organised tuberculous tissue held

patent by extracavitary forces. It is likely that both these elements were present in the composition of this cavity. It is known that the cavity was formed in an area of consolidation which would give rise to a considerable amount of organised diseased tissue in and around its walls. In the films of 13.12.46, the cavity was not distended to any marked degree. It cannot be certain that the cavity was not distended at another time. Lack of marked distension of the cavity might be explained in two ways: firstly, owing to the amount of disease around the cavity the surrounding tissues would not be resilient; secondly, the cavity may have been governed by a bronchial movement valve (p.112, 117). Endobronchial disease, which forms the commonest basis for such a valve mechanism, was seen on bronchoscopy. If the valve was of the common type which traps air distally on expiration before the air has finished passing proximally, but releases during the following inspiration any undue positive pressure which might have resulted from an act such as coughing, a small mean or static positive pressure should result which would keep the cavity inflated. On the other hand, if as is suggested later, the cavity was inflated at occasional intervals, this would accord with a more marked degree of swelling of the bronchial mucosa, leading to an occasional

marked positive internal pressure which would be re-:lieved mainly by absorption of air: the cavity walls would be prevented from much distension owing to the nature of their pathology and that of the surrounding tissues.

It is not necessary to call upon a check valve due to enlarged hilar glands, of the type shown in figure III (2), to help explain the persistent cavity. Furthermore, had such been present it is reasonable to suppose that further collapse by means of the phrenic paralysis and pneumoperitoneum would most likely have caused massive atelectasis in the lower lobe; which did not occur.

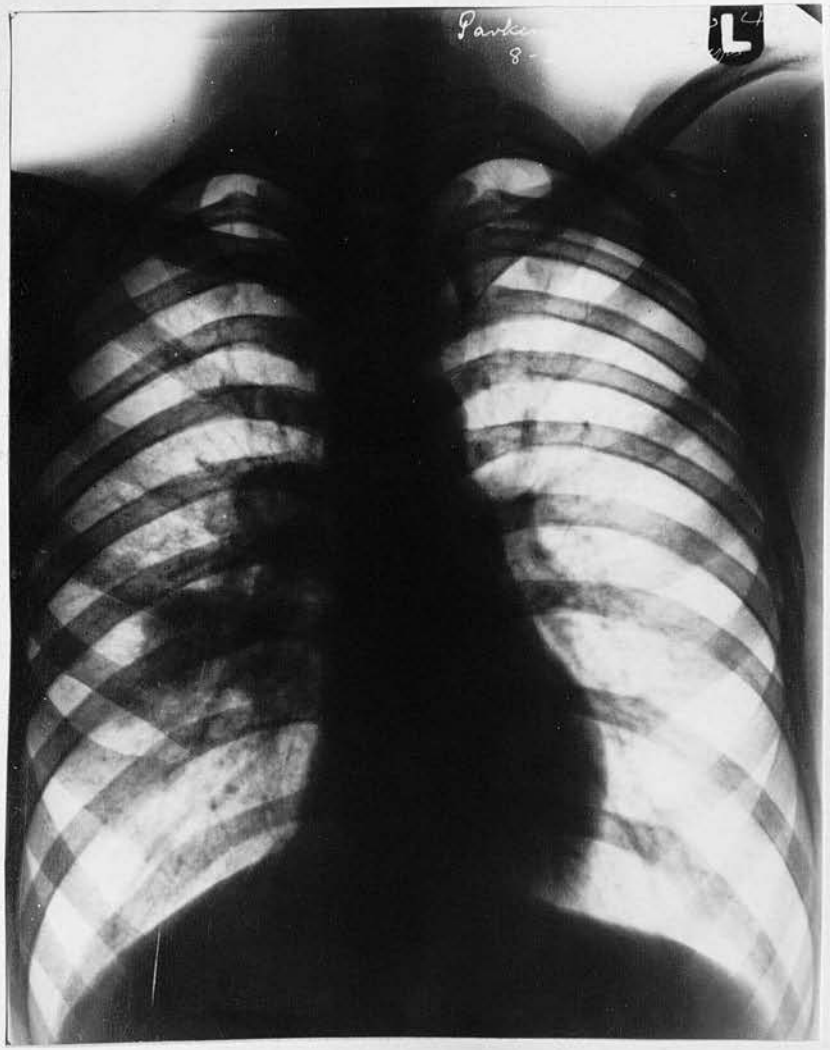
The question remains: why did not the pneumothorax close the cavity when apparently the phrenic paralysis did? The answer is probably that the pneumothorax produced satisfactory collapse from a lateral direction; the rise of the diaphragm supplied the necessary relaxation in a vertical direction: the combined collapse was instrumental in producing the necessary relaxation of the cavity area to cause occlusion of the draining bronchus. Before considering more closely the manner in which the cavity closed, there are some features related to the new relaxation produced which require examination. The pronounced triangular shaped shadow in the right cardio-phrenic angle is probably due to the scoliosis.

The slightly pronounced infundibular heart shadow suggests that the heart was a little rotated, the right ventricle being rotated slightly to the left. This rotation might have been produced by the scoliosis. A second point is that judging by the X-ray films, especially the left oblique film of 13.12.46, there may have been some adherence of the lung to the diaphragm near the lower mediastinum: if not to the diaphragm, the nature of the lesion would lead to the expectation that adhesions might have formed to the lower mediastinum. It would be wrong, however, to read too much into either the appearance of the heart shadow or the suggestive radiological appearance of adhesions. Either of these features, if present, would be suggestive of a retractive force between the lower lobe and the lower posterior mediastinum. But phrenic paralysis, and even pneumoperitoneum, are not likely to have produced much vertical relaxation of the mediastinum: a little might have been produced by the pneumoperitoneum but whether enough to have been effective in itself is questionable. Setting aside all these speculations it can only be concluded that the raised diaphragm, in conjunction with the pneumothorax produced a reduction in the extracavitary forces with a resultant hilarwards relaxation of the cavity area and the draining bronchus. If the lower

lobe was more or less fixed to the immobile lower mediastinum, the relaxation must have followed even more than usual the direction of a lever, fixed to the mediastinum and rotated clockwise towards the hilum. With relaxation of the cavity walls, and shortening and relaxation of the bronchus, both would be permitted to collapse. It can well be visualised that as the cavity walls relaxed and even contracted a little with the new relaxation of the area, the maximum effect would be likely to fall upon the broncho-cavitary junction which would close. However much or little the final relaxation may have been, the result was probably to convert an intermittent, into a complete, bronchial occlusion. The slight and occasional nature of the positive sputum was indicative of an occasionally open, rather than an intermittently open bronchus. If that was the case the cavity may be conceived as having been open at occasional intervals, not with each inspiration. The absence of any subsequent positive sputum indicates that the bronchus was finally closed and in that manner, rather than as a result of scar tissue contraction within its walls, the cavity may be presumed to have closed in this case.

CONCLUSIONS.

Im this case a cavity was kept patent on account of a bronchial movement valve. This valve had probably assumed a more marked degree of stenosis so that opening was at intervals rather than with each inspiration. By raising the diaphragm in conjunction with the pneumothorax the necessary amount of relaxation was afforded to produce complete bronchial occlusion, probably at or near the broncho-cavitary junction.



(8·2·46)

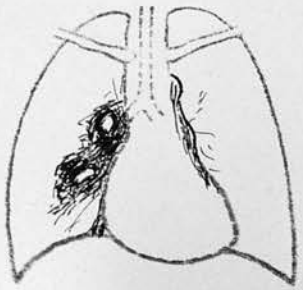
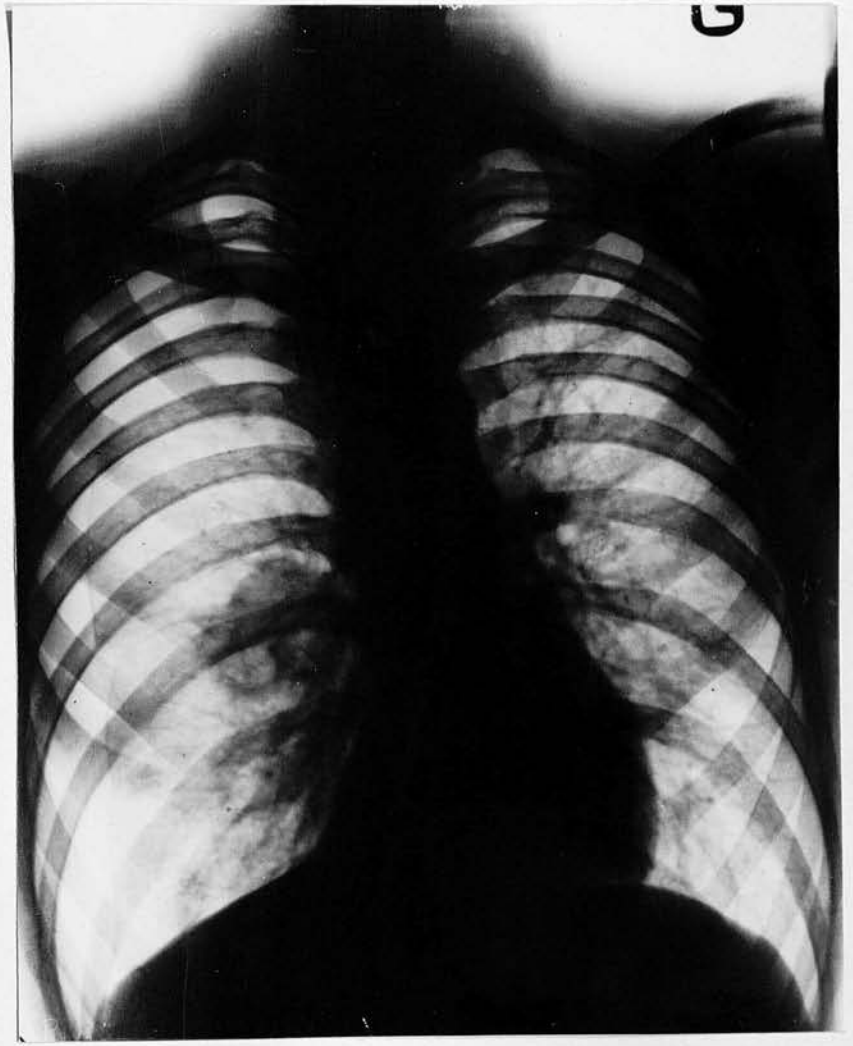


PLATE I

Case 1.



(13·12·46)

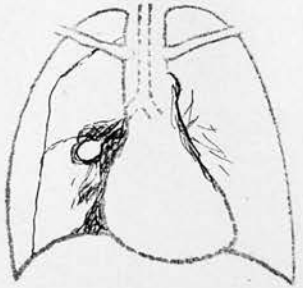
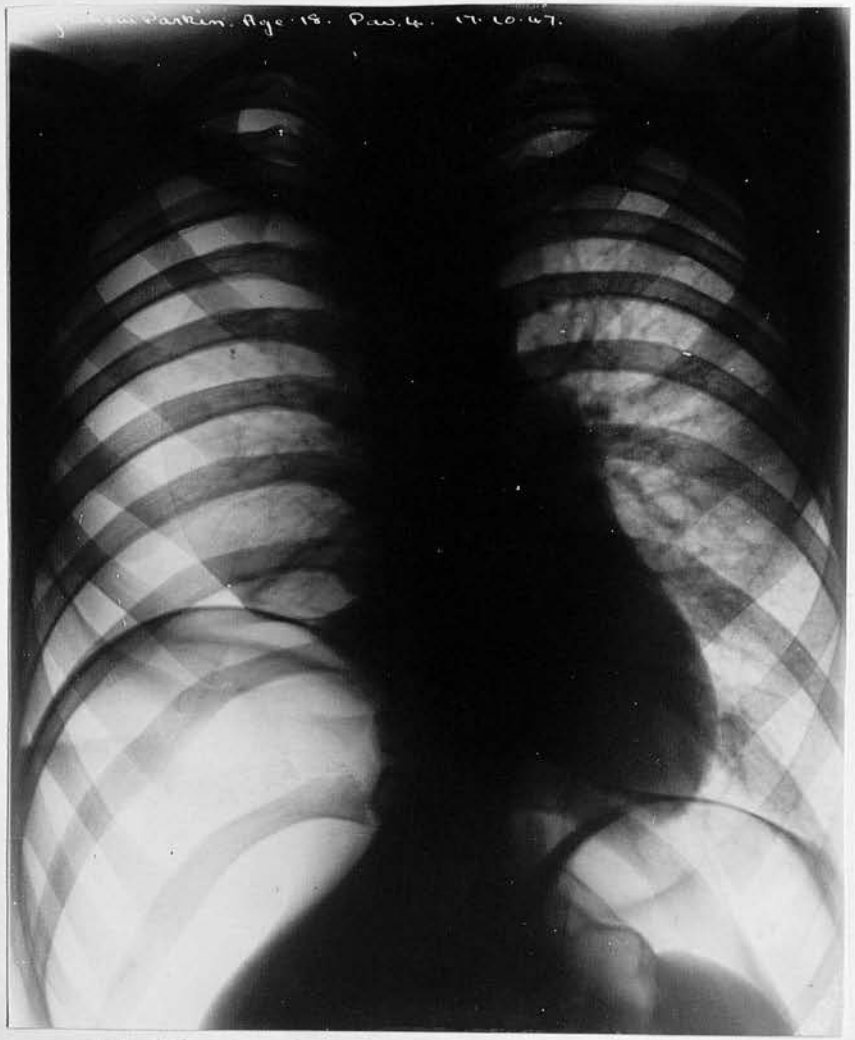
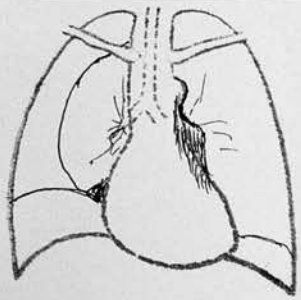


PLATE II



(17.10.47)



Case 1

PLATE III

Case II.

Name. S.B. Age 28 Occupation. Moulder.

Admitted.10th April, 1945. Discharged 14th May,1948.

History on admission.

Cough 2 months ago and some pain the right shoulder. Otherwise always healthy. Has not lost weight. Now has a slight cough and sputum, the latter T.B. positive on admission.

Progress Notes.

Pre-admission X-ray. (22.3.45) Heart and mediastinum drawn slightly to the right but no sharp definition of trachea. Right lung. A fairly homogeneous shadow at the apex but no definite cavitation seen. Slight scattered infiltration in mid and lower zones. Left lung. Doubtful infiltration mid and lower zones.

X-ray on admission (13.4.45) (Plate IV) shows a circumscribed homogeneous density at the right apex. Appearance suggestive of a cavity irregularly "punched out" of this density at the level of the clavicle anteriorly and the upper border of the 3rd rib posteriorly.

(25.4.45)Right artificial pneumothorax induced.

(11.5.45) X-ray shows heart and mediastinum now central. Right artificial pneumothorax collapse visible with apical adhesions. Definite cavity showing at the level of the

first rib anteriorly. Cavity appears elongated and has no wall apart from the circumscribed area of density at the apex. The apical density mainly has the appearance of a hardening lesion which has not as yet appreciably displaced the upper mediastinum.

(11.7.45) X-ray shows cavity still elongated vertically, irregular in shape with no suggestion of dis-:tension.

(20.8.45) X-ray shows cavity about three times its former size, measuring $\frac{7}{8}$" x $\frac{5}{8}$", oblong, more regular in shape and appears distended. At the same time there is to be noted an increased density near the lower pole of the cavity in the region where it might be expected to find the broncho-cavitary junction. This density has the appearance of an increased local tuberculous reaction with some atelectasis. The commencement of this process can be detected in the previous film (11.7.45).

(21.9.45) Little change in radiological appearances except cavity a trifle smaller: shape fairly regular and no cavity walls of its own.

(30.10.45) Thoracoscopy. A complicated mass of apical adhesions present mainly in the region of the subclavian artery. Main mass of

adhesions sectioned leaving some close to mediastinum.

(6.11.45) On X-ray, lung appears adherent to the superior mediastinum at a level just above the upper border of the clavicle. Cavity larger than ever, reaching its maximum size ($\frac{7}{8}$" diameter) and appears more spherical. There is an appearance resembling dilatation of the bronchus near the lower pole of the cavity. The general appearances suggest a cavity distended by a bronchial check valve mechanism and surrounded by a mixture of fibrotic, pneumonic and atelectatic lung tissue (see diagram, Figure VI).

(6.12.45) (Plate V) Cavity a trifle smaller. Dense mass still present at lower pole of cavity but appears to have hardened a good deal.

(24.1.46) X-ray shows: <u>Right lung.</u> Pneumothorax collapse increased: signs of some generalised atelectasis. Cavity no longer appears distended; is much smaller and elongated. <u>Left lung.</u> Infiltration mid and lower zones is now definite.

(1.2.46) Pneumothorax collapse increased still further: upper lobe becoming relatively airless. Cavity still present, a little distended and slightly bigger.

(7.3.46) Second thoracoscopy considered as an attempt to deal with the mediastinal adhesions. This thought inadvisable by thoracic surgeon.

(29.3.46) Right phrenic crush performed to supplement pneumothorax in an endeavour to produce a more satisfactory relaxation to close the cavity.

(5.4.46) Pneumoperitoneum induction

Sputum at this time T.B. negative. Previous-:ly sputum had been T.B. positive but these earlier reports were unreliable.

(7.5.46) (Plate VI) Artificial pneumothorax and pneumoperitoneum. Right diaphragm raised to the level of the 4th rib anteriorly, being an approximate, but very variable, rise of $2\frac{1}{2}$". Cavity still patent and spherical in shape.

(25.6.46) Little radiological change.

(3.7.46) Sputum T.B. positive and consistently so until 2.5.47, inclusive.

(8.7.46) In view of the likelihood of right thoraco-:plasty being performed and the uncertain condition of the left lung, it had been decided to induce a left artificial pneumothorax; which was carried out on this date.

(23.7.46) X-ray shows: Right lung. Artificial pneumo-:thorax and pneumoperitoneum maintained.

Right diaphragm has dropped 1". Cavity spherical and slightly bigger; clear cut and no definite wall of its own.

Left lung. Shallow artificial pneumothorax collapse.

(20.8.46) X-ray shows little change. Occasionally of late, slight evidences of a differentiated cavity wall have been detected.

(1.10.46) X-ray shows right artificial pneumothorax pushed once more and the upper lobe again relative airless. Cavity still well patent. The patient's general condition at this time was good, and he was allowed up.

(21.11.46) Thoracoscopy performed for a second time on the right side in an attempt to free the lung from superior mediastinal adhesions. Lung found to be flattened to the superior mediastinum and no further section of adhesions attempted.

(10.12.46) Little radiological change.

(14.2.47) Bronchoscopy. Larynx, trachea and carnia were found to be normal. The right upper lobe bronchus was oedematous, red and slightly narrowed. No definite tuberculous caseation was to be seen. The middle and lower bronchi were fairly normal and so also

were the left bronchi. The bronchoscopist suggested that the positive sputum was coming from the right upper lobe.

(20.5.47) Right artificial pneumothorax was being abandoned.

(Plate VII) X-ray shows pneumothorax practically obliterated on the right. The mediastinum is drawn to the right and is peaked out by fibrous tissue retraction towards the cavity, the tip of the peak being ¼" from the cavity wall. The cavity, which is spherical and unaltered in size, lies at the level of the clavicle. The cavity can now be seen surrounded by a thick, differentiated ring of density: in other words it has a wall of its own and moreover the appearances suggest that it is largely composed of connective tissue. There is still a good deal of dense shadowing at the apex which, from its appearance and the drawing across of the mediastinum, is also largely fibrotic in character. The dense shadow about the lower pole of the cavity has by now completely cleared.

(12.6.47) Sputum T.B. negative once more.

Left artificial pneumothorax and pneumo-:peritoneum slowly abandoned prior to thoracoplasty. Last pneumothorax refill

3.7.47; last pneumoperitoneum refill 5.6.47.

(26.7.47) Thoracoplasty (First stage) Upper 3 ribs removed. "A very difficult apicolysis because of density and chronicity of disease".

(14.8.47) X-ray shows cavity still patent.

(18.8.47) Thoracoplasty (Second stage.) The wound was reopened. Fluid and clot were removed from the Semb space. Large segments were resected from 4th and 5th ribs and less from 6th and 7th ribs.

The patient had rather a stormy postoperative period during which ½ pint of blood was evacuated from the Semb space and secondary suture and excision of a wound sinus was carried out. There was some pyrexia. However, by 1.10.47 patient's condition was reported good, with only a small sinus left at the top of the wound.

(1.29.47). X-ray shows cavity still visible and much the same size.

(18.11.47) Cavity patent on X-ray, spherical in shape, its centre ½" below the level of the lower border of the clavicle.

(20.1.48) (Plate VIII) Cavity still patent; oblong in shape.

(12.2.48) Sputum T.B. positive.

(31.3.48) X-ray shows: Right lung. Cavity probably still present but indefinite. Left lung. Infiltration has almost completely cleared up.

(5.5.48) Sputum T.B. negative.

(14.5.48) Discharged home with cavity patent and uncertain prognosis, but considered unsuitable for further surgical treatment.

There had been some heavy staining of the sputum before discharge, but this soon cleared up.

Throughout the patient's stay in the Sanatorium his general condition had been, on the whole, good. He was ambulant part of the time and at such times looked well. During the first 5 months his temperature had been a little unstable, rising occasionally to 99°F, but thereafter any isolated rise of temperature was probably associated with treatment.

TABLE I /

T A B L E 1.

To show the position of the Cavity

Date	Anterior rib or interspace	Posterior rib or interspace	Distance of cavity centre from general line of edge of mediastinum (in inches)	Remarks
13.4.45	Clavicle (upper border)	3 R	1½	
11.5.45	1 R.	4 I	1¼	After R.A.P. induced.
11.7.45	1 R (lower border)	5 R	1½	
20.8.45	1 I.	5 I	1¾	Cavity distended.
21.9.45	"	"	1½	
6.11.45	"	"	1½	Cavity maximum size.
6.12.45	"	"	1¼	
24.1.46	"	"	1	Cavity smaller. A.P. more collapsed.
1.2.46	"	"	¾	A.P. even more collapsed.
7.5.46	1 R. (lower border)	"	1	After P.P.induction.
25.6.46	"	"	⅞	
23.7.46	"	"	⅞	After L.A.P. induction
20.8.46	1 I	"	⅞	
1.10.46	"	"	¾	A.P. very collapsed. plus P.P.
10.12.46	"	6 R.	¾	
20.5.47	1 R & Clavicle	3 I	¾	(R.A.P. practically obliterated. Mediast. drawn well across to right and peaked towards cavity.
14.8.47	1 I	4 I	¾	After 1st stage Thoracoplasty.
18.11.47	1 R	5 I	¾	After 2nd stage Thoracoplasty.
20.1.48	1 R	5 R	1	

Reference to column 3 shows clearly the progressive migration of the cavity towards the mediastinum, being drawn by fibrous tissue retract-:ion. To allow for variations in positioning of the patient and in diaphragm levels the most satisfactory method of estimating the position of a cavity for comparison in a series of X-rays was considered to be recording the level of the cavity relative to the anterior and posterior rib (R) or interspace (I). Measurements from the centre of the cavity to the edge of the mediastinum cannot be accurate on account of the difficulty in defining the edge of the mediastinum at times. Measurement to the mid line does not allow for mediastinal shift.

SUMMARY

In this case a patient who was in fairly good general condition had a largely fibrotic lesion at the apex of the right lung which contained a cavity. Both the cavity and much of the fibrosis were in the early stages of development when first the disease was detected. Artificial pneumothorax and partial division of adhesions failed to collapse the cavity and the apex was seen to be firmly adherent to the superior mediastinum. Three months prior to the division of the adhesions there were radiological evidence of a localised exacerbation of the tuberculous disease just below the cavity in the region which was most likely surrounding the draining bronchus. Shortly after the commencement of this exacerbation the cavity became distended to three times its former size. Adhesion section merely increased this distension. The active area near the lower pole of the cavity commenced to heal, however, and six months from the onset of this activity, during a period when the artificial pneumothorax was being pushed to a marked degree of collapse, the cavity ceased to be distended and became smaller. Phrenic paralysis and pneumoperitoneum proved to be useless. Left artificial pneumothorax was induced in preparation for the now inevitable right thoracoplasty, and the right artificial pneumothorax was eventually abandoned.

Bronchoscopy showed inflammatory swelling of the mucosa of the right upper lobe bronchus. Left artificial pneumothorax and pneumoperitoneum were maintained until shortly before the thoracoplasty was performed. In spite of a seven rib thoracoplasty the cavity still persisted at the time of the patient's discharge from the Sanatorium. Formerly irregular in shape, the cavity became smooth and regular in outline, but it was only after the cavity had been watched for over a year that any sign of a differen-:tiated cavity wall could be detected. This eventually (20.5.47) showed up as composed of a moderately thick connective tissue and there were signs of considerable retraction of fibrous tissue in the area between the cavity and the mediastinum. From start to finish the sputum was alternately positive or negative for varying periods with nothing apparently significant about the changes.

DISCUSSION

A number of lessons may be learnt from this case. A demonstration has been given of the employ-:ment of various means to supplement an ineffective artificial pneumothorax, such as phrenic paralysis with pneumoperitoneum, and increasing the pneumothorax collapse to the point of approaching atelectasis of a whole lobe. In this case all such means proved to be

equally futile and a great deal of time was wasted. In addition the work of a subsequent thoracoplasty in closing the cavity was in all probability made more difficult owing to increasing rigidity of the walls of the cavity (see p. 156).

Reference to Table I will show that the cavity was not lifted appreciably following pneumoperi-:toneum and thus the strain was not taken off the superior mediastinal adhesions. It is true there was a chance that the procedure might have so altered the mechanics of the draining bronchus as to have caused complete occlusion but in this particular case, in view of the fibrous nature of the lesion and, it follows, of the cavity walls, an attempt in this way was not really justifiable.

There were probably three factors responsible for maintaining the patency of this cavity: one main factor and two of lesser importance. One of these factors was mainly incidental and temporary, taking the form of a check valve mechanism which at one stage blew up the cavity to three times its former size. This was associated with the localised exacerbation of the disease in the region of the lower pole of the cavity where the broncho-cavitary junction might be presumed to have been. The most likely happening was that the draining bronchus, quite possibly already partly stenosed by mucosal oedema and peribronchial fibrosis, became yet further occluded by increased

inflammatory swelling on becoming incorporated in the pneumonic exacerbation in that area. Thus what had perhaps been a patent bronchus or a bronchus functioning as a bronchial movement valve (Type I p.112) reached a stage of advanced occlusion. This state is associated with that type of valve mechanism in which the bronchus remains blocked throughout both expiration and inspiration but opens at intervels during coughing or deep inspiration to admit small quantities of air; these remain trapped in the cavity and are absorbed slowly by the blood stream. The cavity being surrounded by connective tissue - being in fact a localised excavation within a partially fibrotic apex - would be prevented from distending to any great extent. The reduction in size again of the cavity was associated, it would seem, with the subsidence of the inflammatory process rather than with the increased collapse of the lung. This supports the contention that this was a bronchial movement valve of the type described. It is more than likely, in view of the bronchoscopic findings, that the bronchial movement valve continued to function, but not in so marked a degree as formerly, causing a smaller positive pressure to exist within the cavity. This, however, was not in itself the main reason for persistence of the cavity.

The main mechanism responsible for maintaining the patency of the cavity was undoubtedly an

extracavitary force, or forces, due to retraction of fibrous tissue in the neighbourhood of the cavity, which was anchored to the superior mediastinum. The existence of connective tissue was manifest by the radiological appearance of the apex and eventually of the cavity wall, and the operative findings when performing the thoracoplasty. The presence of fibrous connective tissue was manifest by the tissue retraction, leading to displacement of the mediastinum. The most significant feature of Table I is the evidence that the cavity was drawn gradually and progressively towards the edge of the mediastinum. This fact, coupled with the fibrotic peaking of the tissues at the border of the mediastinum further demonstrates that there was a good deal of fibrous connective tissue near the superior mediastinum - as throughout the apex in general - to which the apex of the lung was firmly adherent. Thus the cavity was situated between two opposing forces which tended to pull its walls apart: the retraction of the fibrous tissue near the mediastinum above and the elastic pull of the bronchi and pulmonary tissues anchored to the root of the lung below.

The third contributary factor was the rigidity of the cavity walls. If the cavity could have been seen pathologically later on in its history, it would most likely have been seen to possess a fairly smooth lining and moderately thick and rigid walls.

As has already been indicated, a state of intermittent bronchial occlusion might have persisted, at least until the thoracoplasty was performed. Whether this was the case or not, that the draining bronchus was patent is evident from the fact that the sputum continued to be positive. Had the bronchus become completely closed it is questionable whether this cavity would have closed unless after a long time. In addition to the fibrous tissue retraction pulling the cavity walls apart, the cavity walls and pericavitary tissues were composed largely of connective tissue which, unlike normal parenchyma, would have been incapable of compensatory emphysema. Had a strong negative intracavitary pressure been created owing to total occlusion of the bronchus, the tension within the cavity would have had rigid uninflatable, as opposed to spongey inflatable, tissues upon which to draw for the purpose of filling in the cavity space. The strong extracavitary forces would have hindered the progressive coming together of the cavity walls. The effect upon the air trapped in the cavity under such circumstances would be that it would cease to be absorbed until further contraction of the cavity walls produced a less negative pressure (p. 29).

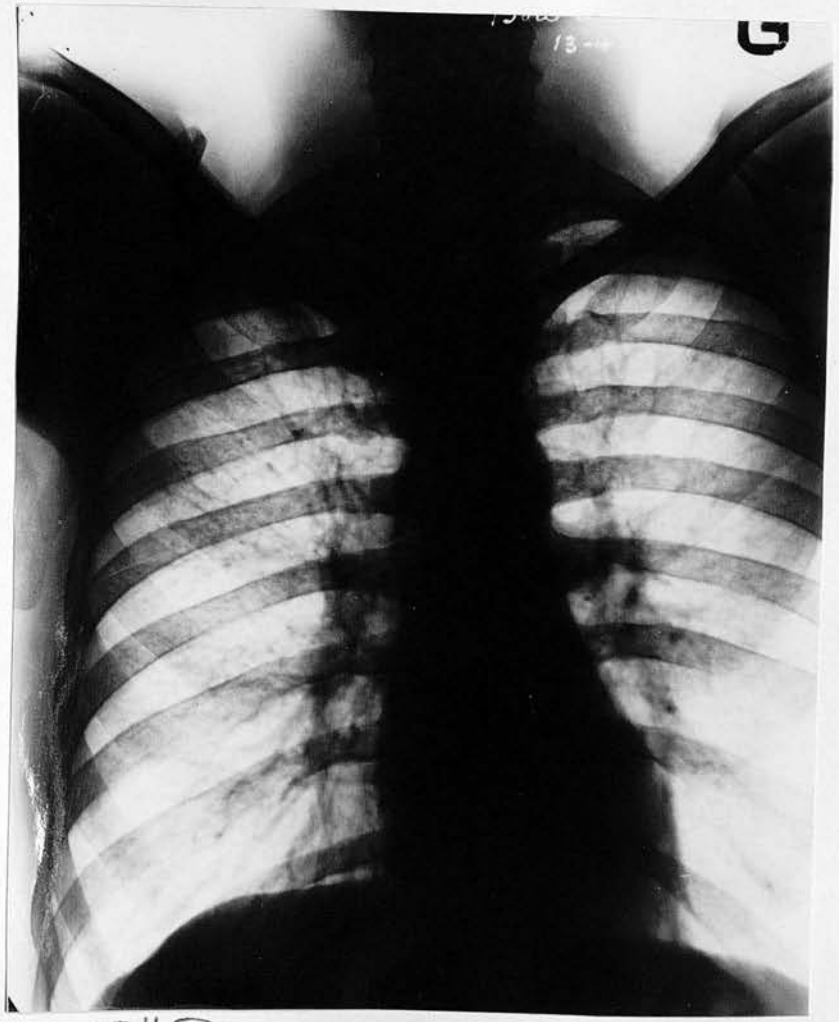
With collapse treatment the cavity was dropped from the level of the 3rd posterior rib to 5th posterior interspace. The portion of the lung, however, in the

region of the superior mediastinum received relatively little relaxation in a hilar direction. The result at the cavity area was a partial relaxation which cannot have been sufficient for cavity closure. Had it been possible to free the apex of the lung from the superior mediastinum and drop it towards the hilum, the cavity would have been permitted to close for two reasons: (1) the extracavitary forces would have been removed thus permitting concentric scar tissue contraction of the cavity walls and (2) the draining bronchus could have become completely closed. The second factor might have been brought about by shortening and contraction of the bronchus or as a direct result of the first factor - contraction of the cavity walls about the broncho-cavitary junction which might have caused complete closure of the orifice. Rigidity of the walls would have acted as a brake to the speed at which the cavity lumen would have become obliterated.

Looking back over the history of this case it is clear that treatment should have been different. When the cavity failed to close with artificial pneumothorax and indivisible mediastinal adhesions were noted, a thoracoplasty should have been performed as soon as the exacerbation of disease in the right lung and the condition of the left lung permitted.

Even with a left artificial pneumothorax the patient would probably have been saved a year of treatment and there would have been a better ultimate prognosis with regard to the cavity. Some would have treated this case by thoracoplasty from the beginning.

The prognosis is very uncertain. The cavity might never close but remain a source of chronic infection to which the patient sooner or later would most likely succumb. The cavity, on the other hand, might heal biologically in which case the out-look would be less grave but still poor. The present hope is that slow progressive fibrous tissue shrinking of the cavity walls and the draining bronchus near the cavity, will ultimately result in permanent blocking of the cavity if not in occlusion of its lumen. If the cavity shows no sign of closing and sputum is positive, open healing might be aided by streptomycin. No surgical approach to the problem of the mediastinal adhesions seems practicable in this case on account of their chronic fibrotic nature.



(13·4·45)

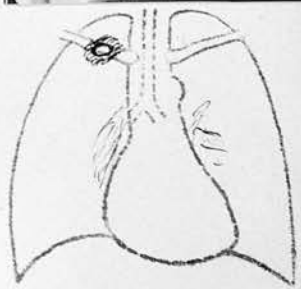
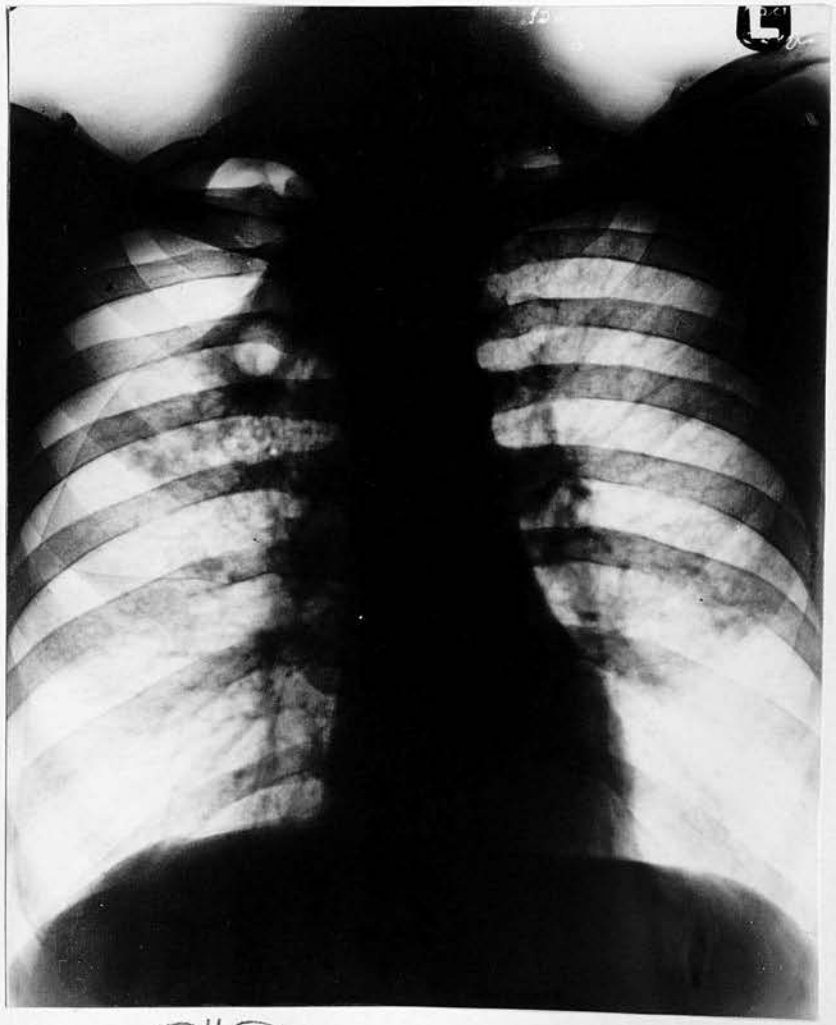


PLATE IV



(6·12·45)

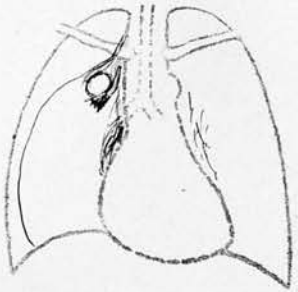
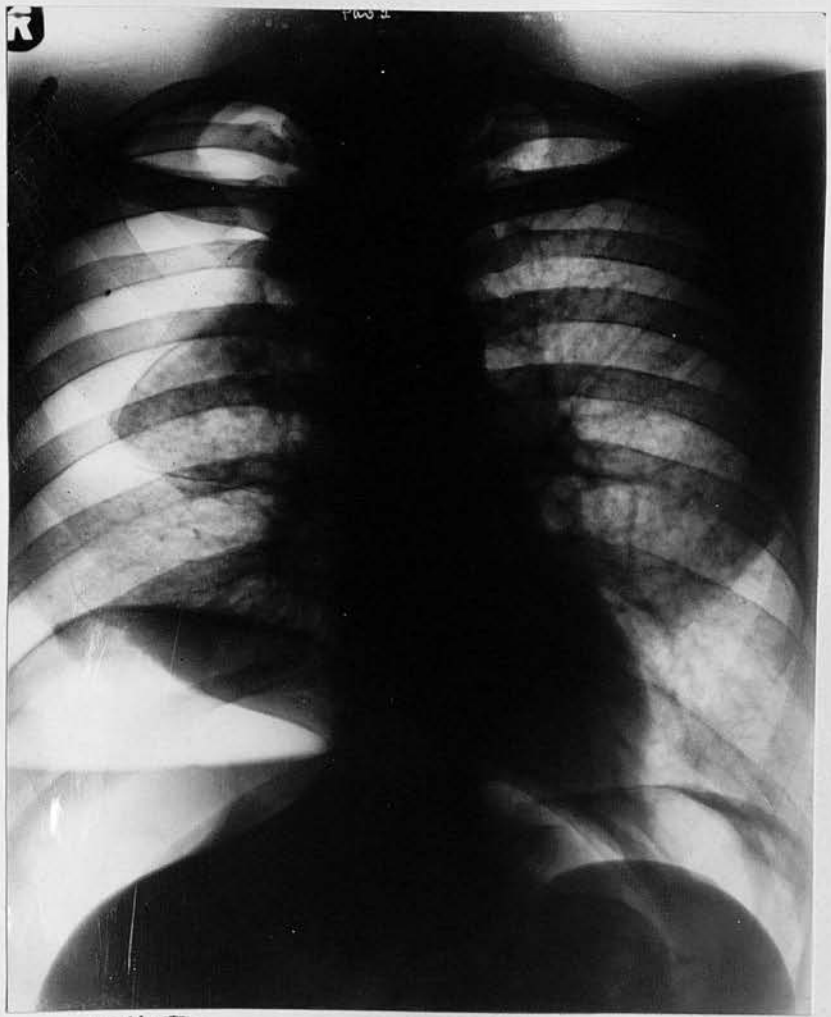


PLATE V

Case II



(7·5·46)

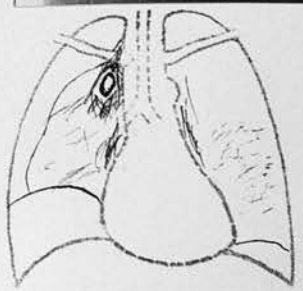
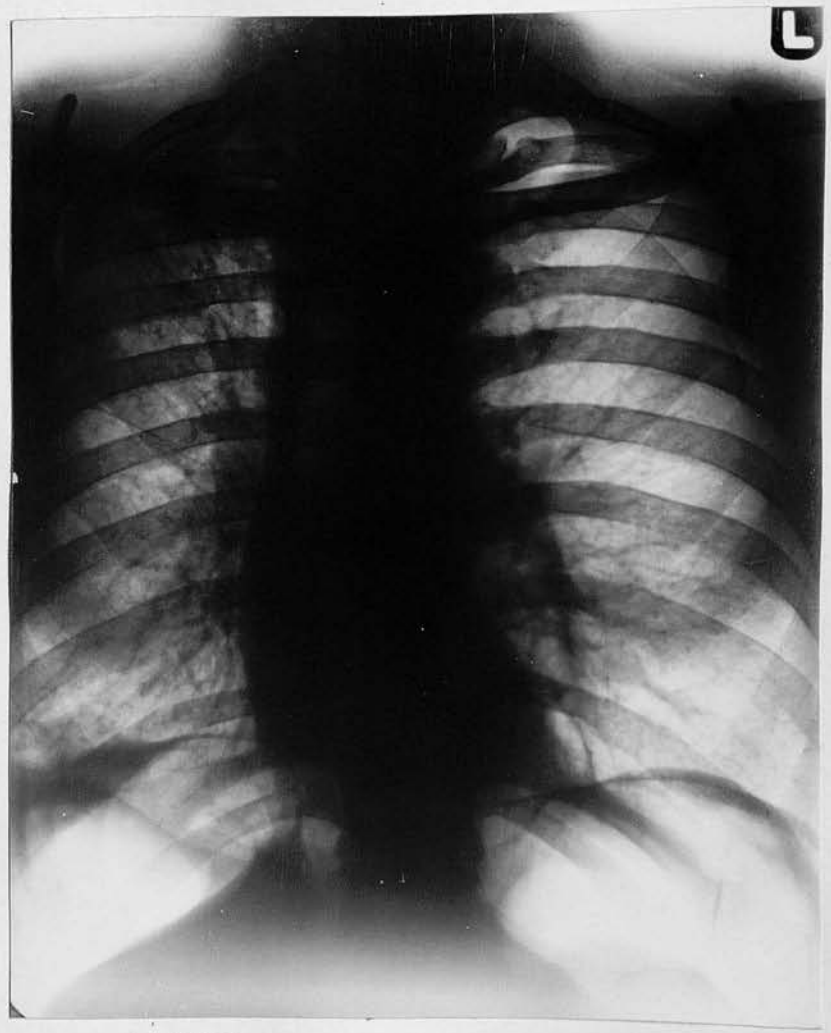


PLATE VI



(20·5·47)

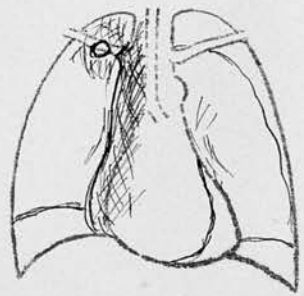
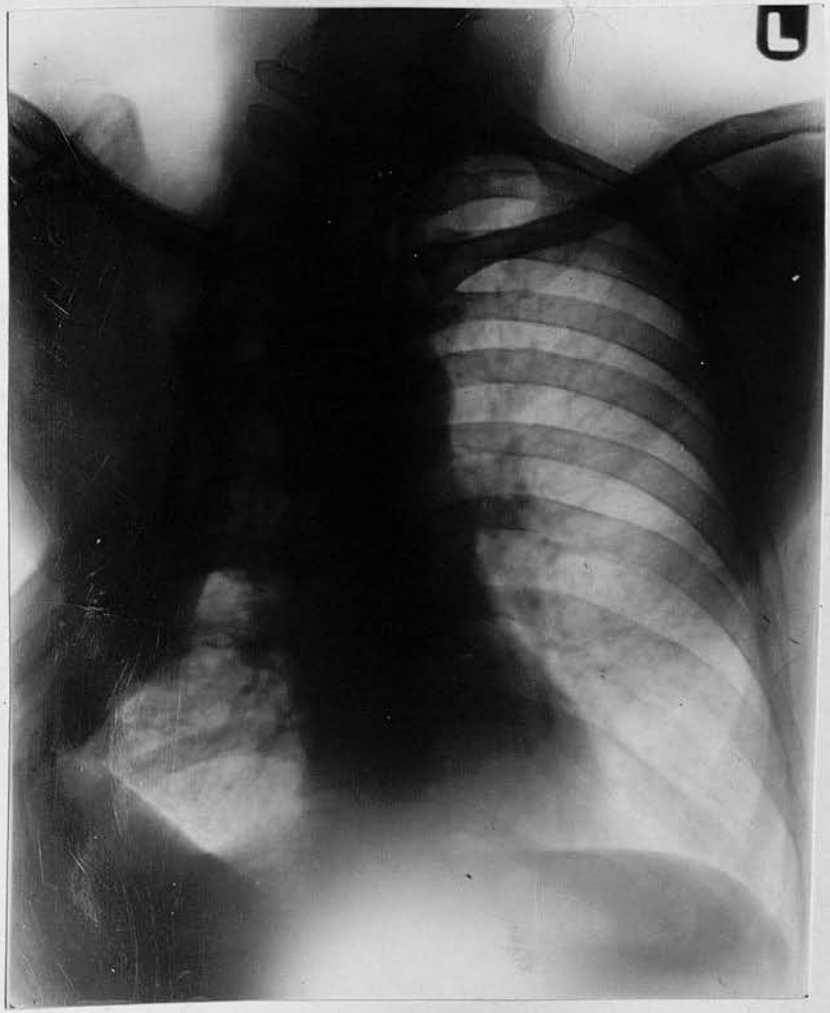


PLATE VII

Case II



(20.1.48)

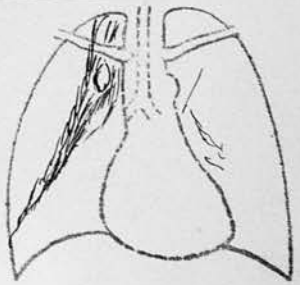


PLATE VIII

Case II

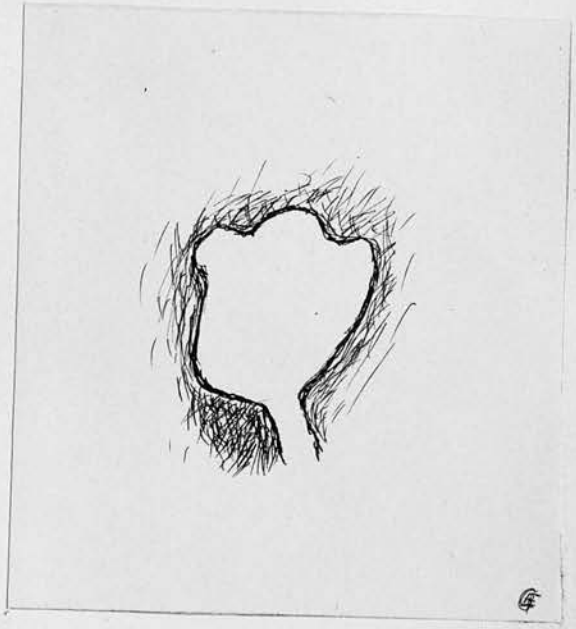


Diagram of cavity appearance 6.11.45.

Figure VI

Case III.

Name. A.S. Age 19. Occupation Ex-R.A.M.C. Orderly.

Admitted. 22nd August 1944. Discharged 10th July 1945

6th September 1945 21st December 1947

Family history. None of significance.

Previous illnesses. None of significance.

History on first admission.

Some pain right apex region and haemoptysis four months prior to admission. No sputum.

During this first period in the Sanatorium the patient was ambulant most of the time. Temperature was unsteady, however, with occasional rises to 99°F. and frequently just above normal.

Preadmission X-ray. (10.7.44) Heart and mediastinum. normal.

Right lung. Infiltration upper zone and probably commencing cavitation behind the clavicle at the level of the 1st anterior rib and the 5th posterior interspace. Im this X-ray and a number of the succeeding ones the right root shadow is pronounced, with increased bronchial shadows showing as linear markings radiating from the hilum towards the apex, interspersed with a little patchy infiltration. Left lung. No definite infiltration seen.

X-ray on admission. (29.8.44). Right lung. Infiltration at apex, moderately hard in character, containing a circumscribed area of density in which

early cavity formation is detected. Left lung.
No change.

(10.10.44) X-ray shows circumscribed area of density at right apex about the size of a florin, containing a cavity.

(23.11.44) (Plate IX). Circumscribed density a little smaller, containing in its centre a cavity which is a little more than ¼" in diameter and which lies at the level of 1st interspace anteriorly and 5th rib posteriorly. The walls of the cavity are composed of the reactive tissue of the area of density; the appearance being that of a thick-walled cavity.

(9.1.45). Radiologically the cavity now presents a clear cut, punched out appearance, slightly oval in shape and surrounded by a thick negative halo - like ring the morbid histology of which might be assumed to be reactive and atelectatic tissue. The cavity conforms partially to Pinner Type I (p. 133) and Ornstein types I and II (p 68). Size 2/8" x 4/8".

(10.1.45) Right artificial pneumothorax induced.

(31.1.45) X-ray shows right artificial pneumothorax with a good deal of collapse of the upper lobe which is adherent at its extreme apex and to the upper mediastinum.

Cavity has dropped to the level of the 2nd rib anteriorly and 6th rib posteriorly but appears unchanged.

(21.3.45) (Plate X). Cavity becoming slowly larger, still at the same level, clear cut, slightly oval and surrounded by negative halo of density. Moderate pneumothorax collapse.

(16.5.45). X-ray show cavity about three times its former size (see Table II), spherical and possessing a dense annular shadow for a wall: it lies at the same level in the thorax. The single line of a bronchus is definable. The pneumothorax is a little shallower than in the previous film.

(10.7.45). Discharged home to await section of adhesions. Patient too restless to remain in Sanatorium. General condition described as fairly good at this time.

(13.7.45) X-ray shows cavity about half its former size. Pneumothorax collapse increased and satisfactory in amount. A very thin inner cavity wall detected, being differentiable from the ring of density which comprises the rest of the cavity wall.

(24.8.45) Radiologically the cavity is bigger than ever before, spherical, with a dense wall.

Marked increase of hilar shadow which presents as a fairly homogeneous density. It is unlikely that this hilar density is simply an appearance due to the collapse of the lung. The lung collapse is shallower once more.

(31.8.45) (Plate XI) Little change.

(3.9.45) Admitted to hospital for adhesion section. This was apparently performed but no note available of details.

(6.9.45). Readmitted to Sanatorium from hospital.

History on readmission.

No cough or sputum, occasional hoarseness. Slight pain in right shoulder. Appetite good, no tiredness nor dyspnoea. Temperature on admission was 99°F. but soon settled to normal.

X-ray on readmission (7.9.45)(Plate XII) The lung has re-expanded considerably following thoracoscopy and the upper lobe now occupies much of the former pleural space above the cavity. The cavity lies at the same level and is the same size. Once again an almost hair-like inner cavity wall can be differentiated, as in the film of 13.7.45. In the light of subsequent findings and radiological appearances the cavity is apparently situated in the base of the upper lobe, posteriorly, subpleurally and adjoining the septum (See Discussion). It is very tempting, however, in

view of radiological appearances to say that the cavity lies in the apex of the lower lobe: this latter can in fact be seen dimpled by the contraction and drag of adhesions and consolidated tissue in the region of the cavity which lies the other side of the septum. The hilar density persists.

(1.10.45) Sputum T.B. positive (Concentration) for the first time.

(1.11.45) (Plate XIII) X-ray shows apparently good collapse from right artificial pneumothorax following adhesion section, and the apex well freed. The appearances, though uncertain, are now those of a horizontally flattened, deflated cavity with thin, irregular shaped walls at the level of 2nd interspace anteriorly and 7th rib posteriorly.

The patient commenced to get up 3 weeks after admission. Following a week-end pass in November 1945 temperature rose to 101·4°F, gradually settling again. On 5.12.45 patient had a haemoptysis and temperature again rose (to 103°F) and took 3 weeks to settle. Patient got up again for 2 hours daily mid January and for most of the time until October 1946.

(3.1.46). (20.2.46) A rarified area seems to appear at the site of the cavity but it is too vague for definition. In the film 3.1.46 the lung is well collapsed.

(2.4.46) (Plate XIV) A cavity shaped like an inverted triangle can be seen at the same level as the rarified area in the preceding two films, (3rd rib anteriorly and 7th rib posteriorly). The cavity measures $\frac{7}{8}$" across the base and $\frac{5}{8}$" perpendicularly from vertex to base. A good deal of circumscribed density surrounds the cavity, suggesting a localised exacerbation of disease. The subpleural position of the cavity is well demonstrated, the base appearing to consist of little else than thickened pleura.

During the same month (April 1946) the temperature was again a little unsteady with a maximum rise to 99·6°F, but returned to normal after a few weeks and remained so.

The pneumothorax collapse was pushed to try to close the cavity, but, probably as a result of the subpleural exacerbation of disease, a pleural effusion developed.

(28.5.46) Pleural effusion shown on X-ray. Cavity smaller.

(18.6.46) (Plate XV)) In X-rays of these dates the
(13.8.46)) cavity appears as a clear cut
(19.9.46) (Plate XVI)) area of rarification in a region of hazy density probably composed of a mixture of inflammatory reaction and

atelectasis. The effusion persists.

Pleural aspirations.

On 15.6.46. 40 cc. clear fluid aspirated. Bacteriological examination of the fluid showed a few mononuclear leucocytes, no acid-fast bacilli or other organisms; culture sterile. Guinea-pig inoculation test negative for tubercle bacilli.

On 20.7.46. 1½ pints of slightly turbid fluid aspirated and gas replaced. Thereafter on four other occasions, up to and including 21.6.47, roughly one pint of fluid was aspirated and gas replaced. The fluid became clear again by the final aspiration.

(5.9.46) Right phrenic crush performed.

(7.10.46) Pneumoperitoneum induction. Bed.

(5.11.46) (Plate XVII) Right diaphragm raised 3½" with a good subdiaphragmatic air space. The position of the cavity has not been appreciably altered however. The lung surrounding the cavity still has the same pneumonic and atelectatic appearance, and the cavity does not appear to possess anything much of a differentiable wall.

(10.12.46) Radiologically, definition of cavity not very clear but appearance suggests the cavity is not distended but flattened and lying subpleurally. A small amount of fluid can be seen above the diaphragm.

It was apparent that the pneumoperitoneum was useless. Bronchoscopy was recommended prior to letting up the pneumothorax with a view to right

thoracoplasty.

Patient commenced to get up again and was getting up for 6 hours within one month.

(14.2.47). Bronchoscopy. Larynx, trachea and carina normal. Right main bronchus was very red, rather oedematous and irritable, the condition extending into the right upper lobe opening which was narrowed and inflamed. Some muco-pus was present but no definite tubercles were seen. The lower lobe and left bronchi all appeared pale and normal. In the opinion of the bronchoscopist, the appearances suggested a definite inflammatory lesion of the right upper lobe bronchi.

(10.6.47) X-ray shows the right lung re-expanding and the faint outline of a cavity.

(21.12.47) Discharged home, thoracoplasty having been refused.

The patient's general condition was good and he was getting up for 8 hours daily. Generally the patient was unable to produce any sputum for examination: only one specimen was definitely found to be T.B. positive.

(12.1.48).(Plate XVIII) Heart and mediastinum dis-:placed to the right. Right diaphragm still raised, but there is no longer any subdiaphragmatic air. Pleura shows some thickening. Right lung. Suggestion of a cavity with possible traces of an irregularly shaped cavity wall: there remains a patch

of hazy density on the medial side of the cavity.

Left lung. No definite infiltration seen.

(5.5.48) Tuberculosis Officer reports that patient's general condition is fair, progress satisfactory but not yet fit for work. Afebrile and no sputum.

(13.9.48) X-ray shows little change beyond appreciable clearing, now that obliteration of the pneumothorax is well established, above the right diaphragm which is still raised. Cavity appears much the same.

(20.10.48) Tuberculosis Officer reports excellent progress. Patient symptomless and no sputum. Recommended for Rehabilitation Course.

TABLE II /

TABLE II

TO SHOW POSITION AND SIZE OF CAVITY.

Date	Ant. rib or inter-space	Post. rib or interspace	Distance of cavity centre from edge of mediastinum (in inches)	Size of cavity (in inches)	Remarks.
29.8.44	1 I	5 R	$1\frac{5}{8}$	Early formation	
10.10.44	-	-	$1\frac{5}{8}$	"	
23.11.44	1 I	5 R	$1\frac{5}{8}$	$\frac{1}{4}$ (just over)	Plate IX
9.1.45	"	"	1 4/8	2/8 x 4/8	
31.1.45	2 R	6 R	$1\frac{1}{8}$	$\frac{3}{8}$ x $\frac{3}{8}$	After A.P. induced.
21.3.45	"	"	1 2/8	3/8 x 4/8	Plate X A.P. collapse moder-:ate.
16.5.45.	"	"	$1\frac{3}{8}$	6/8 x 6/8	A.P. a little shallower.
13.7.45	-	-	1	4/8 x 5/8	A.P. good coll-:apse (increased)
24.8.45	Same level		$1\frac{1}{8}$	$\frac{7}{8}$ x 1	A.P. shallower again.
31.8.45	-	-	$1\frac{1}{8}$	$\frac{7}{8}$ x 1	Plate XI A.P. a little more collapsed.
7.9.45	Same level		1 2/8	$\frac{7}{8}$ x 1	Plate XII A.P. much re-expanded
1.11.45	2 I ?	7 R ?	1 4/8 ?	6/8 x $\frac{3}{8}$?	Plate XIII Cavity definition uncertain.
20.2.46	3 R ?	" ?	1 6/8 ?	?	Cavity definition vague and uncertain.
2.4.46.	"	"	1 6/8	$\frac{7}{8}$ across base $\frac{5}{8}$ vertex - base	Plate XIV Tri-(:angular cavity (definite now.
28.5.46	2R (lower border)	"	$1\frac{1}{8}$	$\frac{3}{8}$ x 4/8	Pleural effusion
18.6.46	2 R	"	?	$\frac{3}{8}$ X 4/8	Plate XV
13.8.46	"	6 I	$1\frac{5}{8}$	6/8 x 6/8?	Expiration film Size of cavity indistinct.
13.8.46	"	"	?	" ?	Inspiration film " "
19.9.46	"	"	$1\frac{5}{8}$	$\frac{5}{8}$ x $\frac{7}{8}$	Plate XVI After Phrenic Crush: still fluid.
5.11.46	" (upper border)	6 R	$1\frac{5}{8}$?	4/8 x $\frac{5}{8}$	PLATE XVII Diaphragm raised with P.P. Heart pushed to rt.
10.12.46	2 R	6 I	$1\frac{5}{8}$	6/8 x $\frac{7}{8}$	Cavity definition not certain: small amount fluid.

(continued)

TABLE II (Continued)

Date	Ant. rib or interspace	Post rib or interspace	Distance of cavity from edge of mediastinum (in inches)	Size of cavity (in inches)	Remarks
10.6.47	2 R	6 R	1 6/8	$\frac{5}{8}$ x $\frac{7}{8}$	Lung re-expand-:ing. Cavity outline faint.
12.1.48	"	5 R	2"	6/8 x 6/8	Plate XVIII. Superior media-:stinum is not displaced as much as lower media-stinum. Cavity indistinct.

SUMMARY

The resistance of this patient to tuberculosis was good. The pulmonary lesion, assessed radiologically, consisted of fairly hard infiltration in the right upper zone in which there was a sharply defined focus of infection the centre of which underwent necrosis and excavation. Based upon radiological appearances and bronchoscopic findings the cavity was placed subpleurally, posteriorly, in the base of the upper lobe close to the interlobar septum. From the earliest available X-ray there have been signs of some peribronchial reaction in the upper zone, probably accompanied by enlargement of the right hilar glands. Bronchoscopy subsequently revealed endobronchial disease in the upper lobe. Six months after the first X-ray, a right artificial pneumothorax was induced (10.1.45). There were apical adhesions and before

these were cut the cavity became inflated (16.5.45). There was no apparent cause for this at the time beyond the radiological prominence of one bronchus which led towards the cavity. Pneumothorax collapse was fairly shallow. Being restless, the patient was discharged home to await adhesion section. After becoming smaller, the cavity inflated again more than ever (24.8.45), three months after the first inflation. Now, however, there is a marked increase in the hilar shadow which remains much the same over the period when the apical adhesions are freed (3.9.45). The patient was readmitted to the Sanatorium following thoracoscopy and the lung collapse was far too shallow. Two months later (1.11.45) the lung was well collapsed and the cavity, though indefinite in outline, appeared to be deflated. Cavity definition became clear once more five months later (2.4.46) when its borders were defined by what appeared to be a localised exacerbation of the tuberculous process. This was followed by a sterile pleural effusion which subsided with aspirations. All this time the cavity persisted above the level of the fluid. Right phrenic crush was performed (5.9.46) supplemented by pneumoperitoneum but was useless as far as cavity closure was concerned. The lung was allowed to re-expand with a view to thoracoplasty but this was eventually refused and the patient went home with the cavity still patent. The patient's condition up-to-date is good. From start to finish the patient

had little or no sputum and at only one period was this found to be positive. The cavity never appeared to possess more than the thinnest trace of a wall of its own; any other so-called wall consisting of the changing pathological state of the surrounding lung tissue. In the light of subsequent views on the nature of the behaviour of the original cavity, the latter may have possessed a greater amount of a more lasting wall structure than otherwise would appear to be the case. The cavity never showed any signs of being inflated again after 7.9.45, four days following adhesion section.

DISCUSSION.

The earlier history of this cavity is not of as much interest as the subsequent story. This might or might not have been a primary lesion with one main focus of infection which subsequently excavated, accompanied by diffuse infiltration in the upper lobe. The age of the patient and the radiological appearance at the hilum support the suggestion that this was a primary complex or, failing that, a superimposed reaction upon a primary focus of infection with root gland involvement. Even when full allowance has been made for the diverse appearances of normal hilar shadows and the radiological changes

produced by collapse of a lung, it must be accepted that the radiological manifestations in the earlier films of this series are those of consolidation of tissue in the right root area which probably consisted of inflammatory exudation mingled with atelectasis. This reaction might have been the sequela of an inflammatory reaction in the hilar glands. The reaction about the root area appears to have had some association with the inflation of the cavity, but the appearances do not suggest that the inflating source was as near the main bronchi as the dense area about the hilum. There was, however, little change to be detected in the appearance of the root shadow at the time when the cavity first was seen to be inflated (16.5.45). (Inflation may have been fairly progressive as signs of enlargement were noted in the film taken two months previously). If the source of cavity inflation was not in the root area it seems reasonable to suppose that inflation was caused by a check valve mechanism within the draining bronchus somewhere in the vicinity of the cavity. This valve condition may have been one of the features present in the embryo stage of a process which later developed and became radiologically evident as the hilar reaction. The bronchial lesion with its valve mechanism may or may not have been responsible for

the hilar condition: both conditions might have developed as a result of a common factor which promoted exacerbation of the disease. That one bronchial shadow was prominent in the film of 16.5.45, when distension of the cavity was evident, might have been fortuitous: on the other hand it might be read as indicating activated disease in the bronchial walls. Bronchoscopy at a later date confirmed the presence of endobronchial disease.

There was little change to be noted in the cavity following the induction of pneumothorax and this treatment does not appear to have been responsible for the cavity inflation. The severing of the pleural adhesions, on the other hand, does appear to have had something to do with the deflation of the cavity. Unfortunately there is a radiologi-:cally blank period between 7.9.45, soon after adhesion section, and 1.11.45 when the cavity was found to be deflated. The positive sputum was found in this interval, incidentally. There is no means of finding out whether the cavity closed slowly or suddenly during those two months. The cavity was unchanged four days after the section of the adhesions: slowness of air absorption following bronchial occlusion cannot be argued here because the cavity persisted. Following adhesion section the valve mechanism may have persisted or, as will be indicated

later, may have become even more active. On the other hand the bronchus may have become temporarily occluded.

During the seven months between 7.9.45. and 2.4.46, on both of which dates cavity outlines are definite, the cavity appears to be irregular in shape and deflated, but the appearances are too indefinite for certainty. Whenin the X-ray of 2.4.46 a cavity is well defined once more, there is a subtle change. The question presents itself whether the same cavity is now being seen? For one thing, the cavity has moved a little further away from the mid line (See Table II) and appears just under the summit of the collapsed edge of the lung. This latter appearance might simply be due to the more established collapse of the surrounding lung bringing the edge in line with the top of the cavity. Also the cavity now assumes a triangular shape with its base to the pleural space. Two possi-:bilities present themselves: one is that this is a subpleural cavity which has not closed; the other is that a new and different cavity is now presenting itself. It becomes necessary, before pursuing the subject any further, to establish as far as possible the location of the cavity.

Originally the cavity lay at the level of the 5th rib posteriorly. According to Brock (13) the upper limit of the oblique fissure is usually at the level of

the 5th rib or interspace. Brock quotes Piersol's description which states that "the fissure of the right lung leaves the vertical column either at the 5th rib or at the interspace above or below it". Thus the cavity if posterior must have lain close to the septum. Bronchoscopy indicated disease of the upper lobe bronchus. This would suggest that the cavity lay at the base of the upper lobe close to the septum. Furthermore, originally the infiltration involved the apex of the lung. Unless it is going to be argued that the infiltration crossed the septum to involve the apex of the lower lobe, it seems more reasonable to accept that the cavity lay in the infiltrated upper lobe. Both before and after the change noted in the appearance and site of the cavity, it can be seen near the surface of the lung. X-ray of 7.9.45 shows dimpling of the surface of what can only be the apex of the lower lobe. It is to be concluded that the septum is fused and the retracted tissues around the cavity pull on the surface of the adjoining lobe and that this is made more pronounced owing to the pneumothorax collapse. In the film dated 2.4.46 the pleura can be seen acting as part of the cavity wall. Tomograph and lateral X-rays would have been of great assistance in placing the cavity: the septum itself might have been distinguish-:able. Unfortunately the only available lateral film

is too faint to be of any assistance. The conclusions are that the cavity lay posteriorly, subpleurally at the base of the upper lobe close to the septum.

Let it be supposed that it is the same cavity which is being followed throughout this history. Following adhesion section, the cavity became deflated. Although the cavity was subpleural it is not likely that the cause of deflation was perforation of the cavity into the pleural space for, had this occurred, the pleural effusion must have occurred earlier on. As it was there was no sign of any effusion five months later, in the film dated 2.4.46. The conclusion must be that the bronchus became occluded temporarily at least. From 2.4.46 the cavity showed more distinct-:ly: on that date there is to be seen a flare up of the disease process around the cavity which, being subpleural, extended to the pleura resulting in an effusion. The cavity thereafter remained patent though never distended, its walls consisting of whatever pathological condition the surrounding parenchyma was undergoing at the particular time - there was no permanent wall apart from a radiologically hair-like inner wall. The cavity persisted because it was a subpleural cavity. Although there was apparently little internal distension - at times the appearances suggest a negative internal pressure rather than a positive - there were no permanent

cavity walls to contribute towards closing the space by scar tissue retraction. In addition, compensatory emphysema of the surrounding lung was deficient (see p.160), part of the cavity wall being composed of pleura: it is not unlikely that this pleura, being thickened, imparted a degree of rigidity to the cavity wall which hindered closure. If the fore-:going conception is to be accepted it will probably be necessary to introduce the supposition that the original cavity extended. If more of the cavity did not become subpleural, at least the cavity seems to have enlarged more in a lateral direction for the centre is further from the mediastinum (Table II). There are two questions which arise before it can be accepted that this was the same cavity which persisted because it was a subpleural cavity: one question is why did not something more in the nature of a permanent cavity wall develop in the wall composed of lung parenchyma? secondly how could such a cavity remain patent in such a locality without showing signs of distension? There is no answer to the first point. To the second question it can only be assumed, in view of the irregular shape of the cavity, that the bronchus admitted air at intervals between which absorption took place, the balance being a mean negative pressure within the cavity. This failed to close the cavity because of factors already mentioned, added to which

was the negative intrapleural pressure. In connection with this last factor, reference to Plate XV and Table I will show how the pleural effusion caused the cavity to become smaller owing to compression produced.

The second possibility is that the cavity of 7.9.45. (Plate XII) is not the cavity of 2.4.46 (Plate XIV). The suggestion presents itself, when looking at the triangular shaped cavity of Plate XIV, that this is an air space within the septum; in other words, the distended, subpleural cavity ruptured into the septum at a point where the latter was not fused and established a new cavity the walls of which were composed of lower lobe pleura below and upper lobe pleura above, the fused septal pleura closing the space peripherally. Upon this hypothesis the interpretation of the radiological series is as follows: In the film of 1.11.45 the cavity has ruptured into the septum where a vague, flattened air pocket is to be seen and which persists as the new cavity. Five months later there is a pleural reaction, both the upper and the lower septal walls of the cavity becoming thickened in consequence (Plate XIV). With the involvement of the base of the triangle, or peripheral wall of the cavity, there is an effusion into the pleural space. There are certain other points which favour

such a view, however unlikely such an hypothesis may seem: there are other points which weigh in the opposite direction. This theory would help to explain the clear cut appearance of the cavity walls with little substance of their own, seeming to be punched out of the lung tissue in many of the later films. It would explain the lateral shift of the cavity which, in the films of 12.1.48 and 13.9.48, persists ⅜" more peripherally from the mediastinal edge than originally. Furthermore the explanation of the dense patch medial to the cavity would be that it is the healed site of the original cavity which had possessed some walls of its own. However attractive such a theory may be, there are numerous objections, one of the foremost being that had such taken place, there was likely to have been an early encysted empyema not an air-containing cavity. The so-called pleural reaction of the cavity walls did not take place until five months after the presumed cavity rupture. Such a cavity would not be likely to persist without pleural symphysis obliterating the space before very long. The bronchial communication with such a cavity would have to be such that the communication with the original cavity was now closed. This would be possible if the draining bronchus lay on the septal side of the original cavity.

The conclusions are that this was a

subpleural cavity which became deflated and then became patent once more. The cavity might have excavated laterally to account for the change of position and it is likely that it became even more subpleurally situated. The dense medial shadow in the latest X-rays was most likely the site of the original part of the cavity, which possessed more permanent walls of its own.

In reviewing the treatment given to this case, the appearance of the subpleural nature of the cavity was a warning of danger. Had cavity closure not been effected speedily the pneumothorax should have been abandoned. The appearance of active subpleural disease in the film of 2.4.46 (Plate XIV) was a warning of inevitable danger and the pneumothorax undoubtedly should have been abandoned forthwith. The phrenic paralysis and pneumoperitoneum were useless for such a cavity which required surface relaxation or compression in order to secure closure.

By 1.11.45 (Plate XIII) treatment appeared to be promising. Having abandoned the pneumothorax however on account of subsequent developments, the next step was either a 7 rib thoracoplasty or an extrapleural pneumothorax. The decision would have been governed largely by whether or not the pneumothorax had been abandoned in time to prevent a pleural effusion, for the extrapleural pneumothorax could not have been

undertaken if the health of the pleura was in question. Apart from this factor the extrapleural pneumothorax was probably the preferable line of treatment. A lateral skiagram would have assisted in placing the cavity for the purpose of such an operation. In view of the postero-lateral situation of the cavity a surgical opinion on this question would govern the final decision regarding the line of treatment to be adopted.

Before a final opinion regarding prognosis was given, tomograph X-rays would be indicated to establish the nature, and even to confirm the presence, of the cavity. Examination of gastric juice or laryngeal swab might reveal the presence of tubercle bacilli in the bronchial secretions which would considerably effect the outlook. The bronchoscopic findings were not compatible with the conception of a T.B. negative sputum at that time. The prognosis depends largely upon whether or not the cavity is patent and whether or not the sputum is T.B. negative. The outlook if there is no cavity and the sputum is T.B. negative is good; otherwise it is the reverse.

At present no further treatment is being contemplated in this case partly because such has been refused and partly because the patient is symptom free. If this could be confirmed with regard to the sputum by more searching means, such a course is

justifiable under conditions of observation. Other-
:wise an attempt should be made to induce the patient to accept surgical treatment such as has been indicated, in order to close the cavity. Failing that, streptomycin, when available for such cases, might produce open healing of the cavity and of the bronchus. Phrenic avulsion would at least help to prevent matters from becoming worse, especially in view of the marked mediastinal displacement, if proper treatment was refused.

(23·11·44)

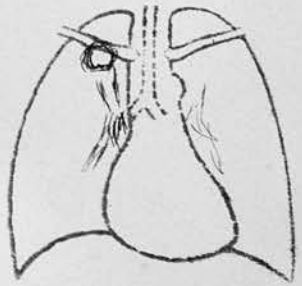
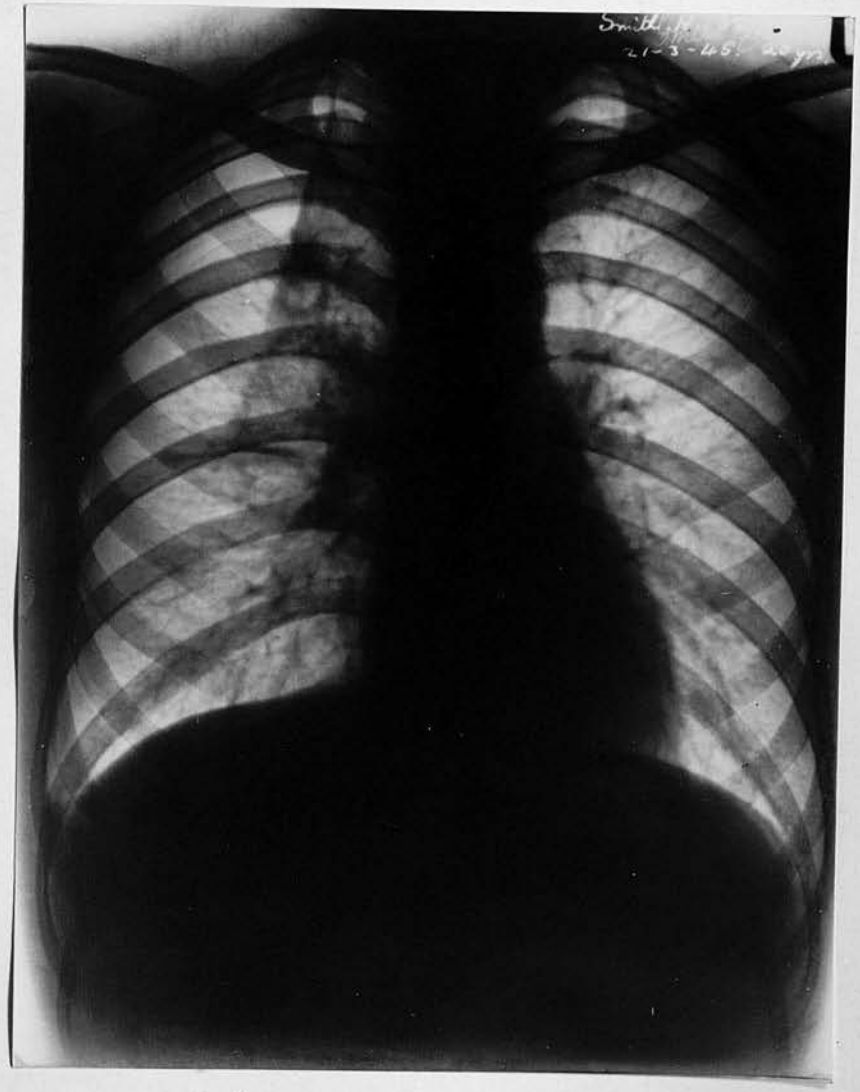


PLATE IX



(21·3·45)

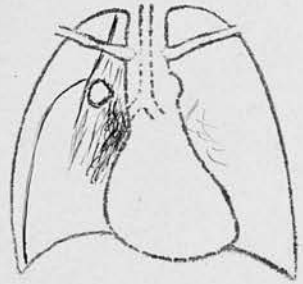
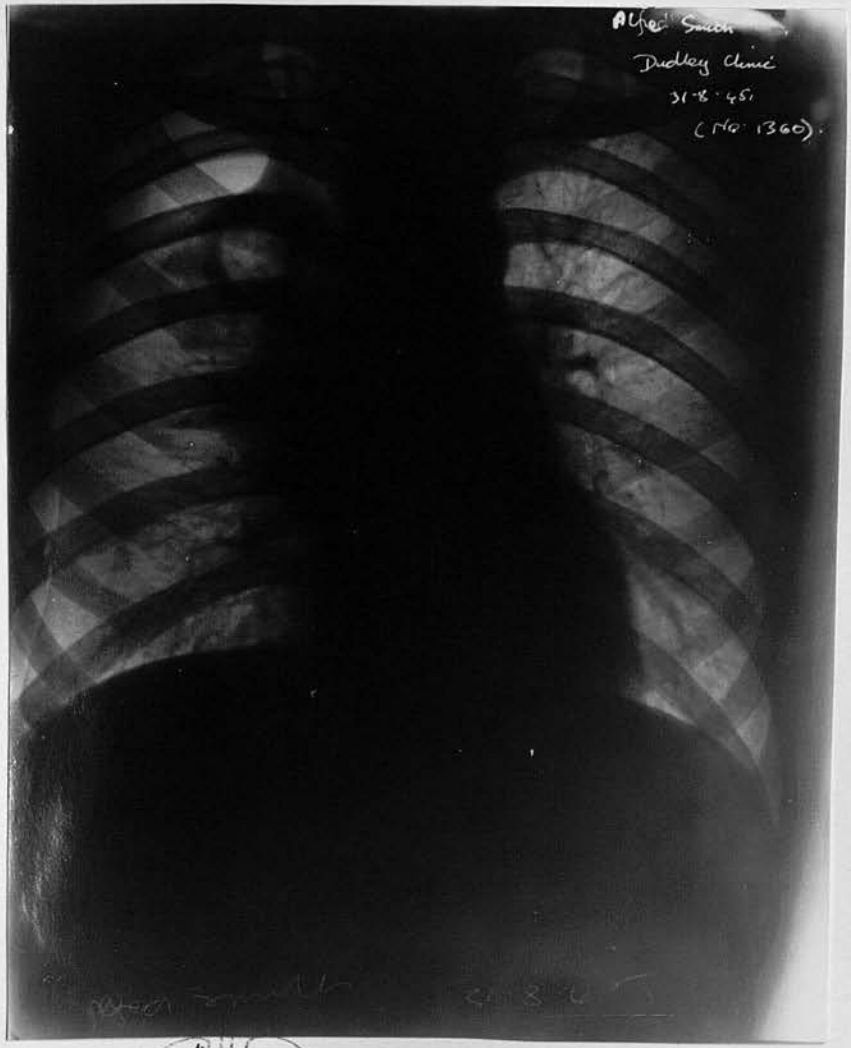
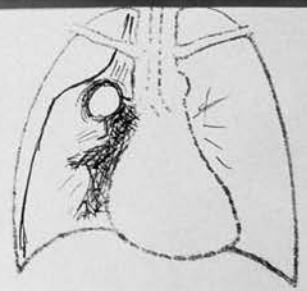


PLATE X

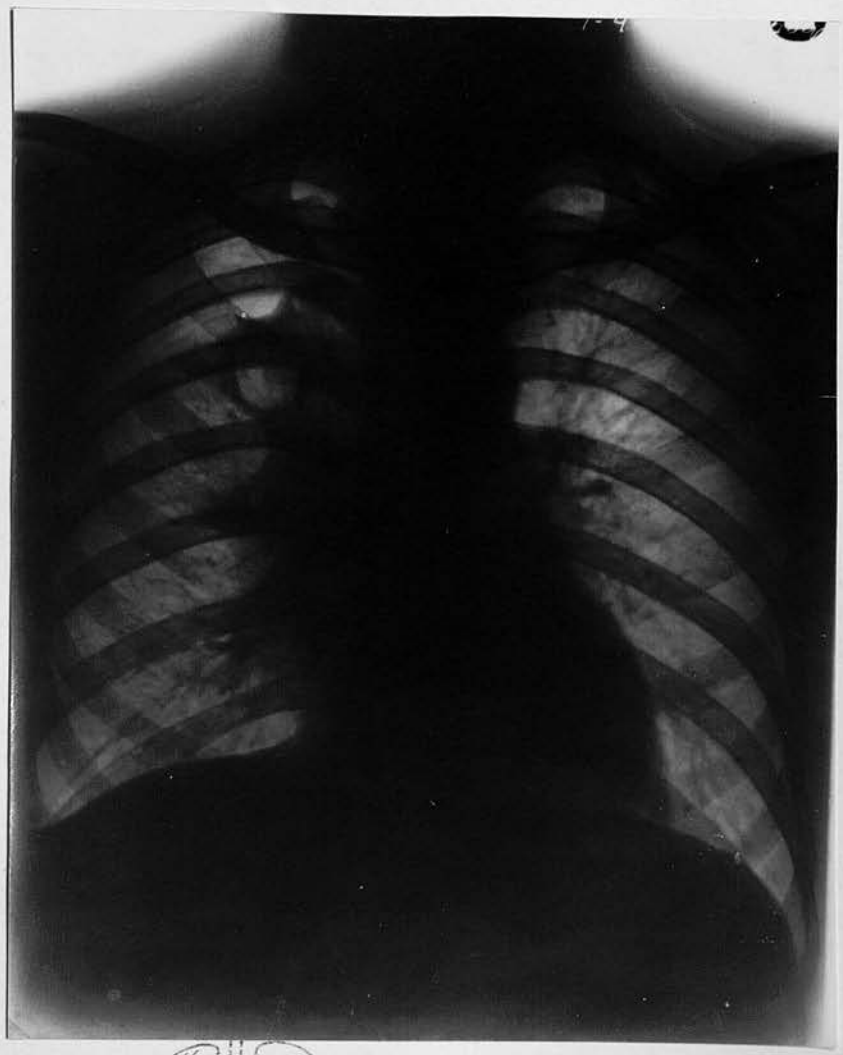
Case III

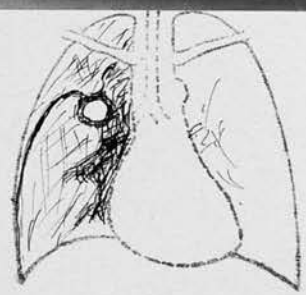




(31·8·45)

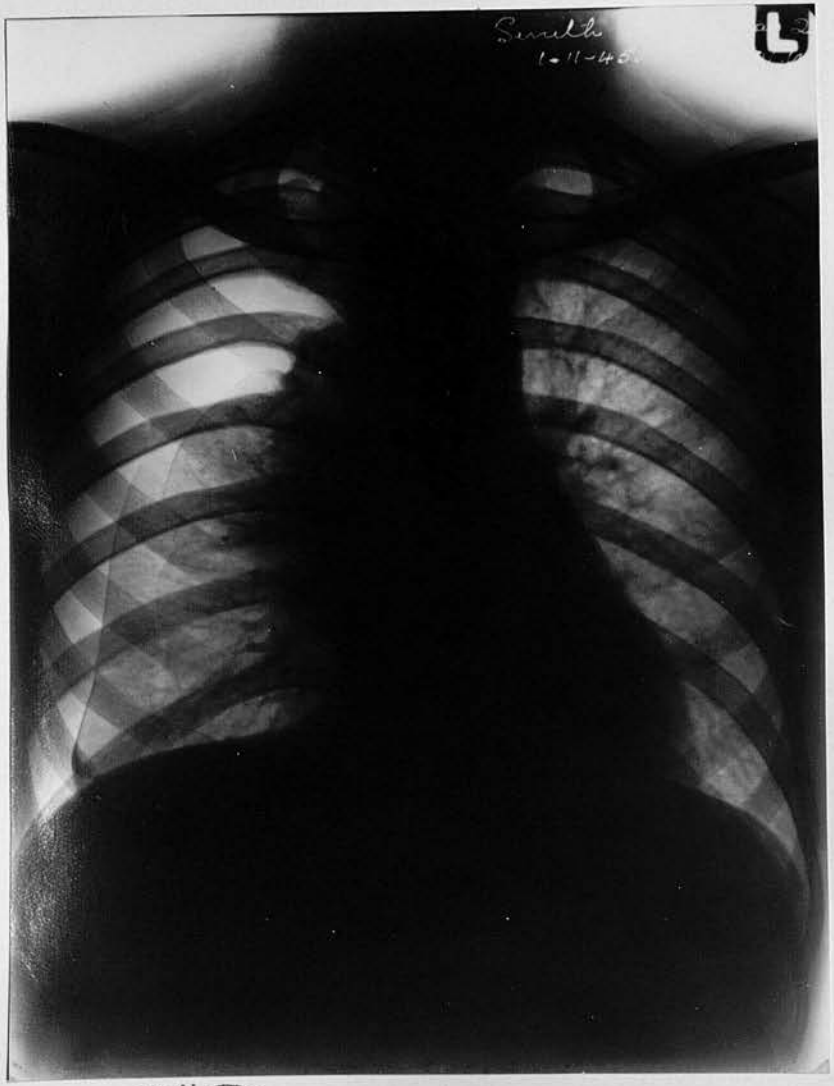
PLATE XI

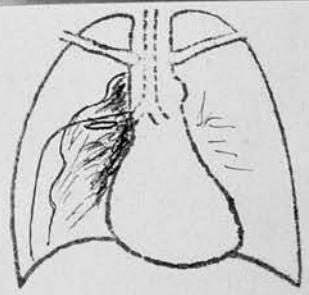




(7·9·45)

PLATE XII

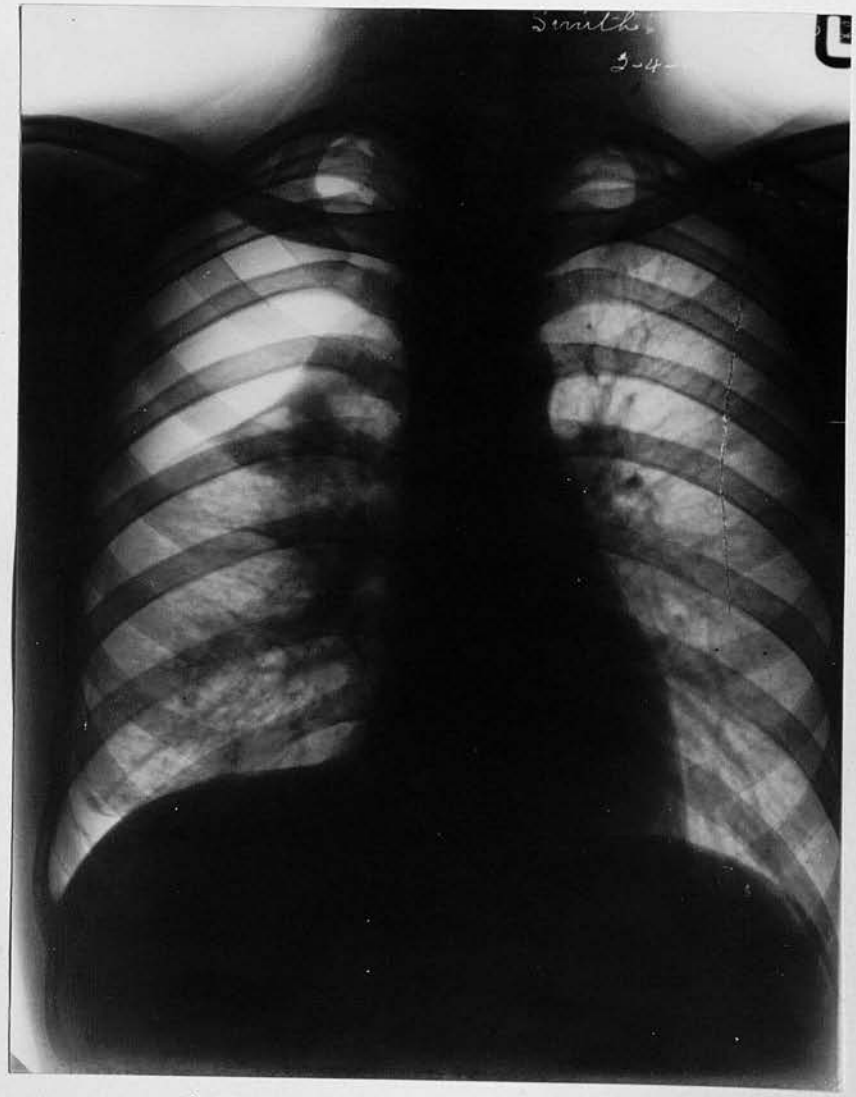


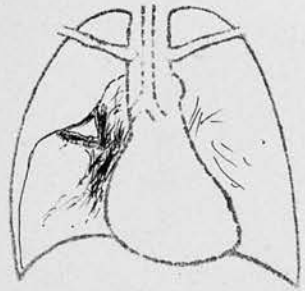


(1·11·45)

Case III

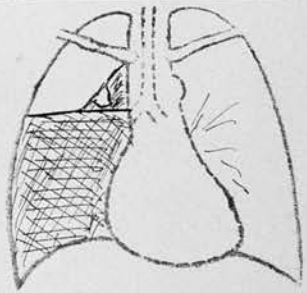
PLATE XIII





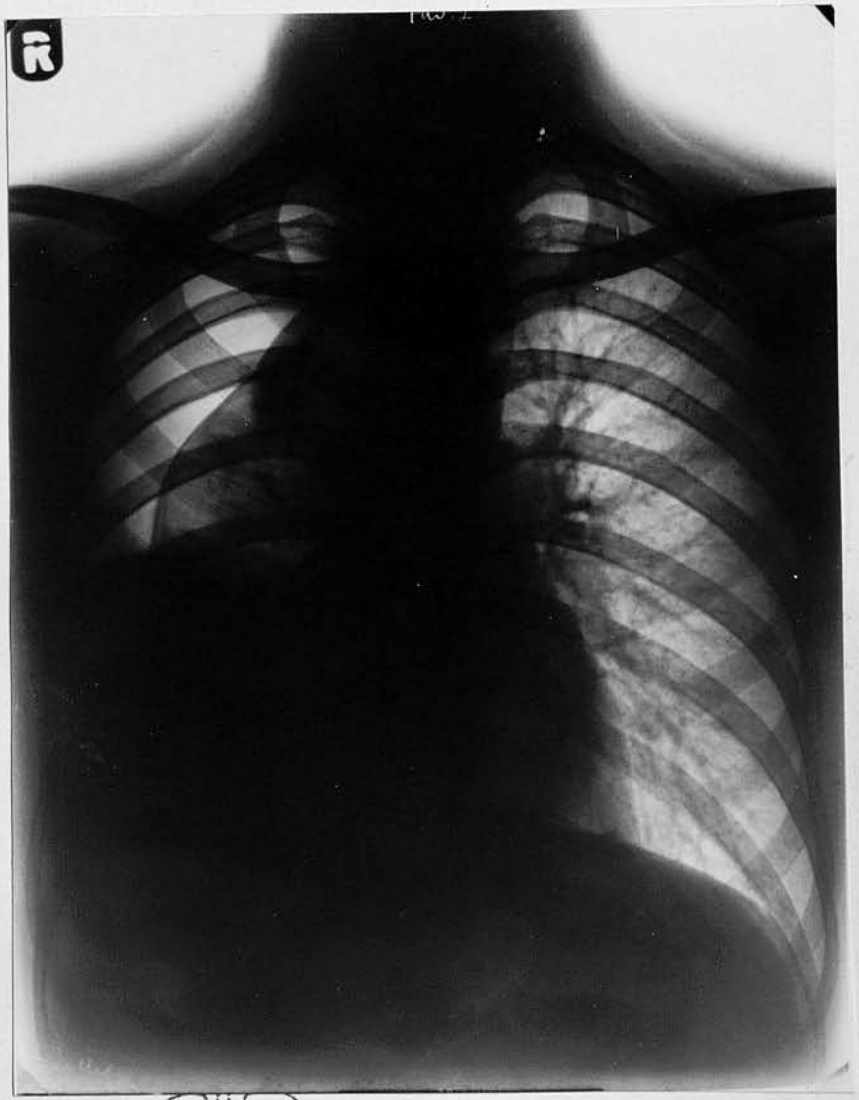
(2·4·46)

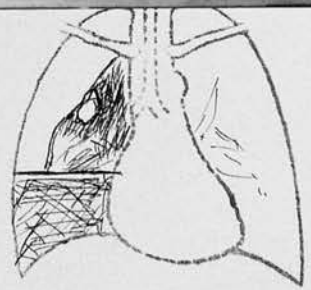
PLATE XIV



(18·6·46)

PLATE XV

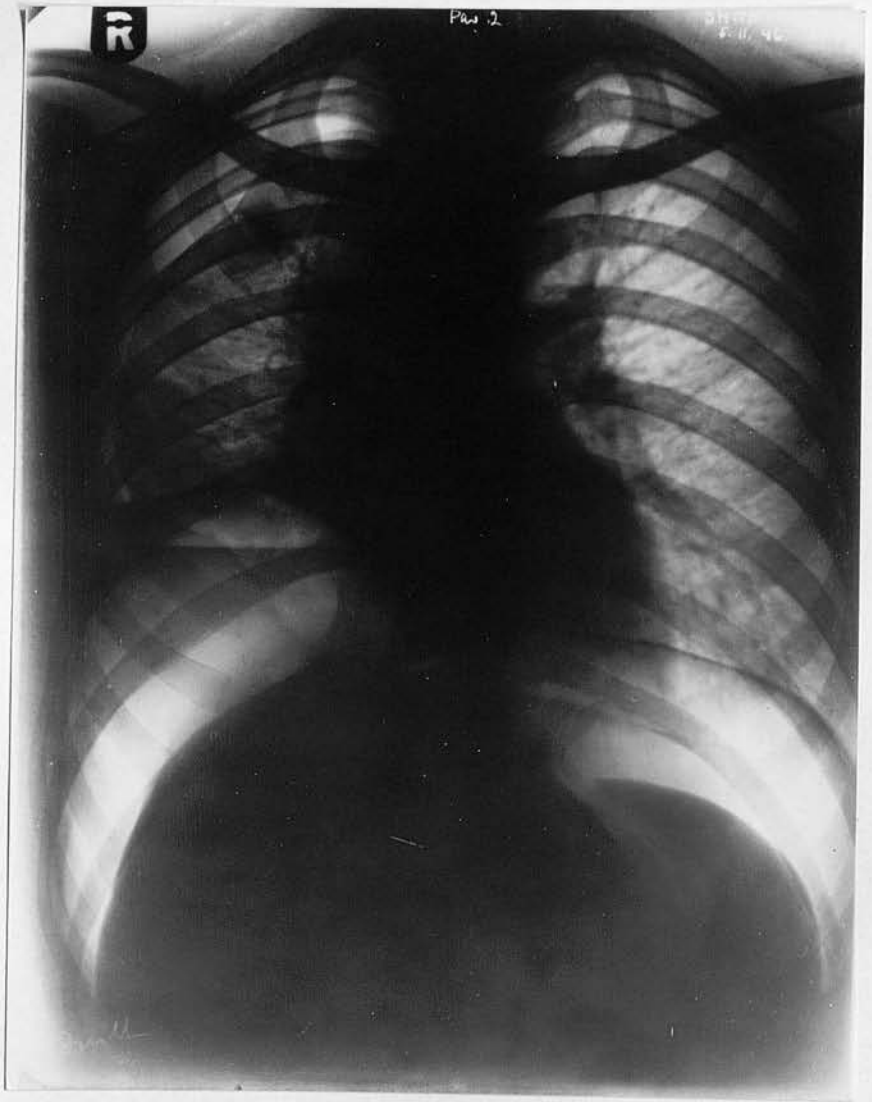


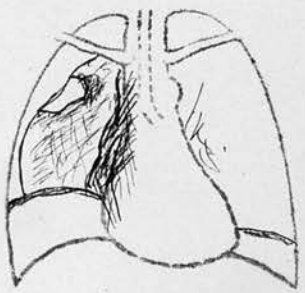


(19·9·46)

PLATE XVI

Case III

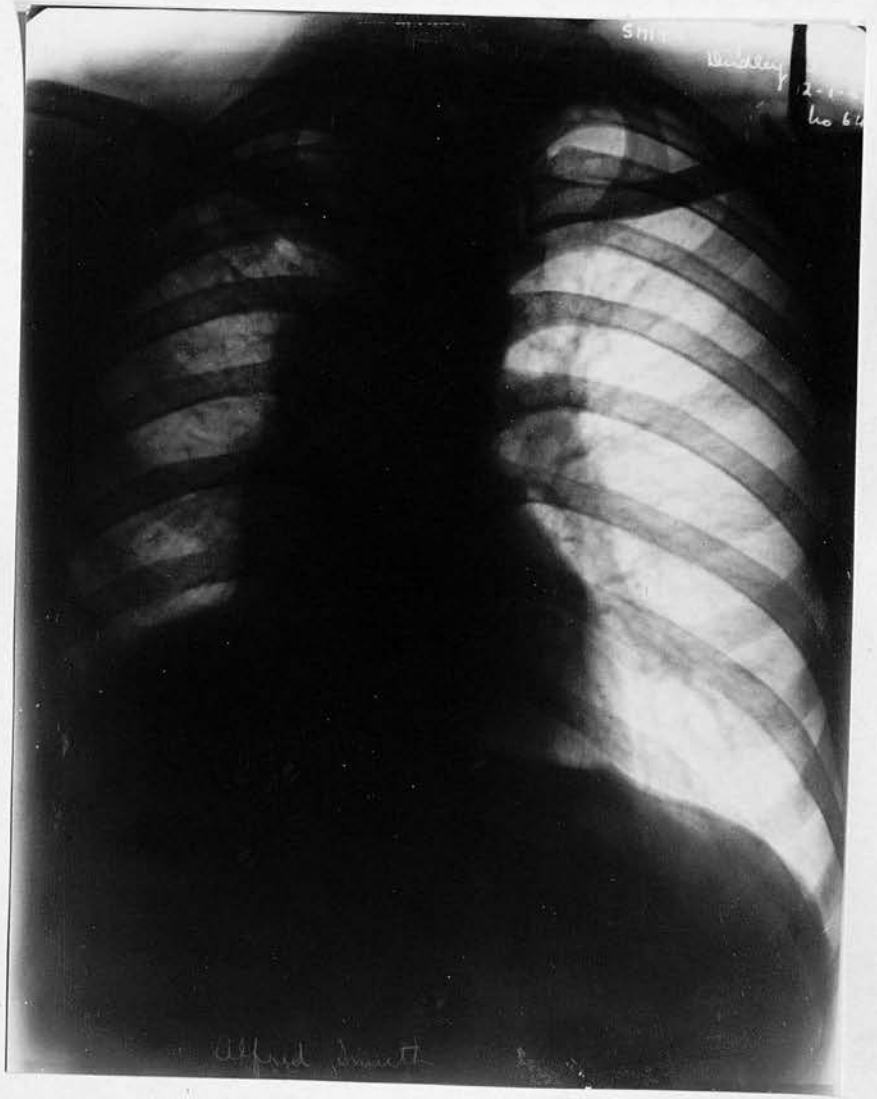


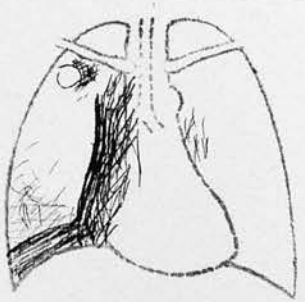


(5·11·46)

Case III

PLATE XVII





(12·1·48)

PLATE XVIII

CASE IV

Name H.O. Age 21 Occupation Bank Clerk. Ex- R.A.F.

Admitted 7th December,1943. Discharged 1st August,1944

25th Aptil, 1946 29th April,1946

23rd July, 1946 14th September, 1947.

Family History: None of tuberculosis.

Past History: No illnesses of note.

History on first admission.

The patient joined the R.A.F. in September 1943. The following month (October 1943) was diagnosed on Mass Radiography as having pulmonary tuberculosis. He was taken off duty and rested. Symptoms at that time (November 1943) were slight and consisted of occasional pain in the upper part of the chest on the left side during the previous six months and occasional lassitude. The patient had no cough or sputum, any sputum obtainable was T.B. negative, and he was afebrile. Weight was above normal. Preadmission X-ray (15.11.43) (Plate XIX). Heart and mediastinum normal. Right lung. No infiltration seen. Left lung. Infiltration upper zone containing a small area of consolidation in which there is a cavity (4/8" x 5/8") slightly irregular in shape and having no walls differentiable from the surrounding pathological process. The cavity is at the level of

the 2nd rib anteriorly and the 4th interspace posteriorly. Two parallel bronchial shadows are prominent, running from the hilum towards the medial portion of the consolidated area and near to the cavity.

X-ray on admission. (10.12.43) Little change, except that the cavity appears more irregular in shape.

(29.12.43) Left artificial pneumothorax induction.

(4.1.44) X-ray shows a shallow pneumothorax. Heart and mediastinum displaced a little to the right. Cavity unchanged.
Patient commenced to get up.

(23.2.44) Pneumothorax very shallow. Cavity larger and appears loculated.

(29.2.44) X-ray shows moderately shallow collapse. Cavity 6/8" x 6/8" in diameter, irregular, loculated appearance, and surrounded by a negative halo of consolidated tissue: suggestion of a fluid level. Two parallel bronchial shadows run towards the medial aspect of the cavity.

(27.4.44) Increased collapse of lung with heart and mediastinum to be seen well over to the right; base of lung well collapsed but mid zone not as much, suggesting lung adherent in this zone. Medial apical adhesions visible. Cavity area indistinct.

(27.7.44) No definite cavity seen and surrounding density shrinking. Patch of opacity to be seen on the lateral base of the lower lobe.

(18.4.44) Discharged home.

During the period whilst in the Sanatorium the patient had maintained his weight, there was no cough and little sputum but the latter was reported T.B. positive on occasions. Temperature occasionally rose to 98·6° - 98.8°F. Weekly refills had been given of 500 - 700 cc. air until May 1944 when refills were reduced to 400 cc. fortnightly. Pressures were negative - zero.

(11.8.44) (Plate XX) Appearance very suggestive of cavitation, surrounded by a small negative halo. Refills 200 - 400 cc. weekly.

(12.10.44) Sputum T.B. negative.

(27.10.44) Radiologically cavity indefinite but appearance suggests less negative halo and more like a cavity wall of its own.

(24.11.44) Little change noted in X-ray appearances. Base of lung collapsed but mid zone still out.

(5.1.45) No definite cavity visible. Heart and mediastinum slightly to the right. Base even more collapsed. Refills 300 - 400 cc. weekly.

(2.3.45) Little radiological change.

(27.4.45) The consolidated area has apparently healed and cannot be seen. There is however a continuance of a "broken" appearance at the site, making cavity closure uncertain.

(15.6.45) (Plate XXI) Little radiological change.

(1.9.45) Sputum T.B. negative.

(7.9.45) Cavity area indistinct. Base even more collapsed and suggestive of some superficial atelectasis at the lateral tip of the base.

(2.11.45) X-ray shows much the same collapse. No definite cavitation seen. Refills continued 500-400 ccs. weekly.

(23.1.46) X-ray much the same, with an indefinite
(6.2.46)
(20.3.46 "broken" appearance at the cavity site.

In February 1946 refills given fortnightly with a view to letting up the pneumothorax. This was apparently a matter of course and not an abandonment.

(10.4.46) (Plate XXII) Pneumothorax shallower. Suggestion of a cavity reappearing. Diaphragm being drawn up.

(25.4.46. to 29.4.46) Temporarily readmitted to Sanatorium for Left Phrenic crush to control re-expanding lung. Screening after operation showed satisfactory phrenic paralysis.

(9.7.46)(Plate XXIII) Artificial pneumothorax obliterated; left diaphragm raised.

Infiltration in all zones of left lung, with a large tension cavity ($1\frac{5}{8}$" x $1\frac{1}{8}$"), centre level with the 1st interspace anteriorly and the 5th rib posteriorly. The cavity possesses a thin wall flattened across the base, and is surrounded by a slight negative halo. Two pairs of bronchial shadows can be seen crossing each other and running towards the cavity.

The notes of the Tuberculosis Officer dated "July 1946" state that the patient had had a relapse during the past four weeks with cough and sputum, a sore throat, loss of appetite and flesh, and lassitude. He was afebrile on the day of that report, with no cough or sputum but dyspnoea noted on exertion.
(11.7.46) Sputum T.B. positive.

(23.7.46) Readmitted to Sanatorium.
Symptoms on admission. Cough and sputum, temperature 98.8°F, night sweats, occasional pain left nipple region. Has lost 6 lb. weight in 6 weeks.

Patient put to bed; allowed "up W.C.".
(24.7.46) X-ray shows cavity smaller (1" x 6/8"), straight lower pole, wall thin and indistinct, except laterally where the wall is denser with atelectasis. Patch of infiltration seen in lower zone near the border of the heart. At the lung base there is pleural thickening

and a small quantity of fluid.

(29.7.46) Sputum T.B. positive.

(1.8.46) Sudden pyrexia 102·6°F. which subsided over nine days and thereafter remained normal.

(21.8.46) Sputum T.B. negative for the next eleven months.

(29.8.46) Reinduction of left artificial pneumothorax attempted: air space found. Given refills of approximately 150 cc. air weekly.

(12.9.46) X-ray shows a shallow collapse in upper and mid zones. No cavity to be seen. The collapse was considered to be selective enough to control the disease temporarily but thoracoplasty recommended as a permanent measure.

Commenced to get up a little soon after this date.

(3.12.46) X-ray shows a little increased pneumothorax collapse, being selective over the upper zone but lung adherent at apex and in its lower half. No cavity to be seen in upper zone.

(30.12.46) Transferred to hospital for Thoracoplasty

(1.1.47) X-ray shows a well pushed down selective collapse. No cavity to be seen upper zone. Bronchial shadows not generally so prominent after this period.

(8.1.47) (Plate XXIV) X-ray shows little change.

(24.1.47) Lung considerably re-expanded but no cavity to be seen in upper zone.

(31.1.47) Pneumothorax collapse shallower. No cavity upper zone.

(8.2.47) Pneumothorax collapse even shallower. No cavity.

(12.2.47) Slight collapse only. No cavity. Left diaphragm well raised (? recent phrenic section).

(18.2.47) Transferred back to Sanatorium. Because no cavity was seen on re-expanding the lung and sputum T.B. negative, thoracoplasty was not considered advisable: phrenic section was performed instead.

The patient was kept under observation, being allowed up 2 hours daily.

(.6.3.47) Radiologically no collapse seen. No cavity. Left diaphragm has dropped somewhat.

(17.5.47) No definite cavity to be seen.

Patient getting up 4 hours daily by this time.

(16.6.47) Right and left oblique X-rays show no definite cavity. Commenced to get up 8 hours daily.

(29.7.47) Sputum T.B. positive on culture.

(14.8.47) Report received, following screening and tomograph X-rays taken at 3", 4" and 5" from the back, to effect that the left

diaphragm was restricted and elevated, its movement only just paradoxical. No definite cavitation was to be seen but there was some basal consolidation. It was suggested the positive sputum might originate from the basal area.

Concerning the basal consolidation mentioned in the above report: for the sake of clarity reference to the lower zone shadows has been mostly omitted. Their exact nature has been very uncertain, areas of consolidation - both atelectatic and pneumonic - with excavation and pleural thickening have all suggested themselves at different times. Tomography supports basal consolidation: lateral films suggest pleural thickening and consolidation. Apart from having been a possible cause of the positive sputum of 29.7.47, these features have not affected the issue and no further reference to them seems necessary.

(15.8.47) Sputum T.B. negative.

(14.9.47) Discharged home at patient's own request, to be kept under observation. Question of thoracoplasty postponed.

Patient was getting up 8 hours daily, had gained nearly 2 stones in weight since admission.

(16.9.47) X-ray shows very doubtful cavitation in upper zone.

(22.3.48) No definite cavity to be seen in upper zone. General condition of patient satisfactory.

(21.10.48)(Plate XXV). No definite cavitation to be seen. Infiltration in former cavity area, like the basal shadow, is harder and the disease processes in the lung in general appear hard and stable. Left diaphragm raised and costo-phrenic angle obliterated. The right lung appears normal. The heart is displaced slightly to the left.

The Tuberculosis Officer reports that the patient is symptomless and has no sputum. Patient has been working regularly for the past five and a half months as a ledger clerk.

SUMMARY.

A unilateral upper zone lesion was diagnosed in October 1943 by Mass Radiography. The earliest available X-ray of one month later showed excavation in a pneumonic area, the cavity conforming to Pinner and to Ornstein, type I. Some bronchial shadows to the area were pronounced. About two months after the diagnosis was made, left artificial pneumothorax was induced. The lung was adherent at the apex and in the mid zone. Thoracoscopy was not carried out. The pneumothorax was maintained for a little over two years with apparent success. Scrutiny of the films

taken over this period, however - and knowing the subsequent history - gives the impression of doubtful cavitation remaining. This became even more suggestive as the lung re-expanded. Phrenic crush was performed (27.4.46) to control the re-expanding lung but two months later the patient had a relapse, sputum became T.B. positive and the cavity was seen to have ballooned out to a large size. The patient was readmitted to the Sanatorium and the cavity was noted to be smaller. Nine days after admission the patient developed a sudden pyrexia which lasted nine days. The left artificial pneumothorax was reinduced and, as it was selective enough to control the cavity area, maintained until thoracoplasty could be performed. Upon re-expanding the lung prior to thoracoplasty no cavity was seen and the sputum was T.B. negative. Consequently the patient was returned to the Sanatorium, after phrenic section had been performed, with a recommendation for observation, thoracoplasty not being considered justifiable under the circumstances. The patient remained in the Sanatorium for a further seven months, under observation, during which time no definite cavity was seen. There was some doubt about this, however, and a positive sputum was detected once on culture. This latter might have been attributable to disease in the lower zone but it is uncertain. More than one year following discharge,

the patient was reported to be well and at work. Although the radiological appearances at that time are still a little dubious, there is no definite cavity to be seen.

DISCUSSION.

Whatever the interpretation of the X-ray appearances during the first period of pneumothorax treatment from the end of 1943 to 1946, subsequent developments revealed that the cavity had not healed: it had remained capable of being inflated to a con-:siderable size. The reason for this failure on the part of the cavity to heal completely was most likely that the artificial pneumothorax collapse was unsatisfactory. The marked collapse of the base of the lung as opposed to the upper and mid zones showed that adhesions were holding up a full selective collapse of the area in which the cavity was situated. Thus the extracavitary forces had not been suffuciently removed for the cavity to heal: by that is meant healed occlusion of the draining bronchus as well as apposition of the cavity walls. Had the draining bronchus closed permanently, the cavity could not have become reinflated as it did in 1946. Although endobronchial tuberculosis had most likely been present at some stage prior to 1946 there is nothing to suggest that this condition or its after effects was at all responsible for the persistence of the

cavity under pneumothorax. Bronchoscopy was not performed but the bronchial shadows strongly suggest that there was or had been a tuberculous reaction within the walls of the draining bronchus - or bronchi, for there were two pronounced bronchial shadows running to the cavity area. It is unlikely there was any active bronchial disease during the later part of the period during which the ineffective pneumothorax was in operation.

During this period the cavity area merely had a "broken" appearance. There was no dense focus of trapped, inspissating caseous contents which would have suggested a marked stenosis and blocking of the draining bronchus. In this case the indications are that the bronchus was patent, and the cavity shrunken and containing little if any caseous matter. The cavity walls were very thin. This fact accounts for the difficulty in detecting the cavity radiologically and also for the changes in size of which the cavity was capable.

Approximately two months after the pneumothorax had obliterated, the cavity was found blown up (9.7.46), the process having been accompanied by a marked constitutional reaction. That this was a tension cavity is clear, not only from examination of the film (Plate XXIII), but from the subsequent behaviour of the cavity which became appreciably smaller again although the lung was not collapsed.

It is certain that the cause for this distension of the cavity was within the draining bronchus. The history at that time, as has been said, is of a systemic reaction. Either there was tonsillitis which caused an exacerbation of the pulmonary tuberculosis or the sore throat was merely coincidental with the lighting up of the tuberculous disease - possibly affecting all zones - which was quite likely precipitated, or made possible by the re-expansion of the lung. Whatever the underlying cause it seems most reasonable to suppose that the endobronchial condition in the proximity of the cavity was aggravated, resulting in swelling of the mucosa and increase of secretions with the result that a check valve was formed. The mechanism might have been more intimately connected with the cavity rather than the bronchus, consisting of pericavitary exacerbation of disease which involved the broncho-cavitary junction causing this to become stenosed and to function as a check valve.

The cavity was already becoming smaller a fortnight after the tension cavity was seen. A reasonable explanation for this fact might be that the general rest had caused a subsidence of the acute reaction. This would result in lessening of the inflammatory oedema and swelling around the bronchial orifice: as a result the valve would become less efficient and eventually cease to function at all.

Within one month the patient had a second sudden relapse with high fever, gradually subsiding over nine days. It cannot be known whether the cavity inflated again at that time as no X-ray was obtained until five weeks after the onset of the febrile relapse by which time a pneumothorax had been reinduced which might have collapsed the cavity again. No cavity was to be seen, however, in that film. It may well be that the broncho-cavitary junction was sealed by the time the second relapse took place. The one and only sputum analysis recorded during this interval, between the onset of fever and the next X-ray, was T.B. negative which, as far as it is worth, militates against a patent cavity.

The question arises as to the part played by the pneumothorax collapse both in the distension and in the subsequent closure of the cavity. Upon re-expanding the lung, the extracavitary forces increased. This would pull the collapsed cavity walls apart. If the cavity is indeed faintly visible on 10.4.46 (Plate XXII) this would account for the reappearance. But the tension cavity was not due to extracavitary forces for the cavity became smaller whilst these forces were unchanged. Whether or not the change in the extracavitary forces when the pneumothorax was abandoned contributed towards the mechanics of the bronchial valve, is another matter.

It is highly probable. Regarding cavity closure: the cavity was already showing signs of becoming smaller before the pneumothorax was reinduced. As has been indicated, it cannot be known for certain whether the penumothorax closed the cavity or whether the result was obtained owing to a continuance of a process already in operation when the cavity was showing signs of becoming smaller: such a process being pathological bronchial occlusion.

It can only be concluded that tuberculous bronchitis - and very likely also an active process in the cavity wall at the broncho-cavitary junction - with or without the aid of the reinduced pneumothorax resulted in apparently permanent occlusion of the draining bronchus; for the cavity remained closed when the lung was once more expanded with a view to thoracoplasty.

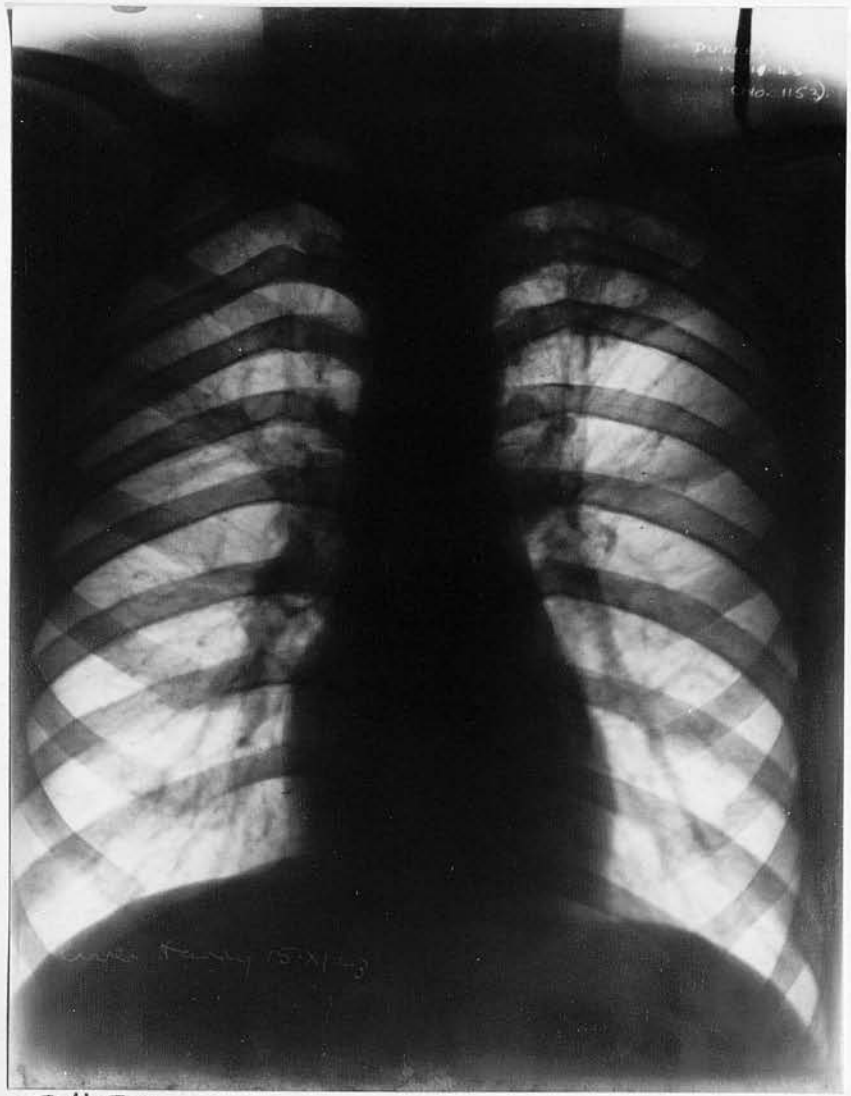
One final objection might be raised: it might be argued that with the cessation of the bronchial check valve, the cavity has subsided to its former shrunken state, the bronchus still remaining open: that this is largely made possible by the reduction in the extracavitary forces due to reinduced pneumothorax and later the phrenic paralysis. It can only be replied that such an hypothesis does not seem at all likely. If this is true it must be argued that the reappearance of the cavity on 10.4.46 (Plate XXII)

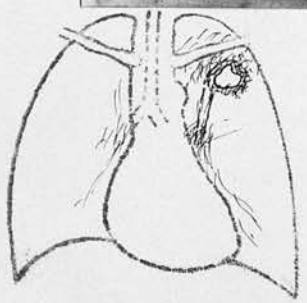
was due entirely to a bronchial valve mechanism and not to extracavitary forces; which seems unlikely.

To conclude: this was a cavity which remained patent under pneumothorax for over two years. After the pneumothorax was abandoned the cavity became a tension cavity owing to an exacerbation of the tuberculous disease which, by stenosing the draining bronchus, created a check valve. This process either with or without the aid of a reinduced pneumothorax finally resulted in permanent bronchial occlusion with closure of the cavity. This has apparently taken place without trapping any appreciable contents, in which case cavity closure must have been rapid. This is compatible with the thinness of the cavity walls. Healing has been by approximation of the cavity walls and there should be a comparatively small scar to mark the site.

In spite of rejecting the suggestion that the cavity has returned to its former shrunken state with a potent bronchus, the radiological appearances leave some doubt as to the true state of affairs at the cavity site. In the film of 22.3.48 there is a long conical shadow with its apex towards the hilum. The sides of the cone are bronchial shadows but the base, which conforms to the original

cavity site, has a suspicious irregular shape. The same but harder appearance is met in the film of 21.10.48 (Plate XXV). In this connection it is pointed out that the tomographs cannot be regarded as offering conclusive evidence of cavity closure. Three cuts, one inch apart, might have missed such a small focus. It must be left as a matter for conflicting opinion whether a thoracoplasty should not have been performed at the time when the patient was submitted for this operation. Time alone will show. One year and eight months after the pneumothorax had obliterated the cavity still appeared to be closed.

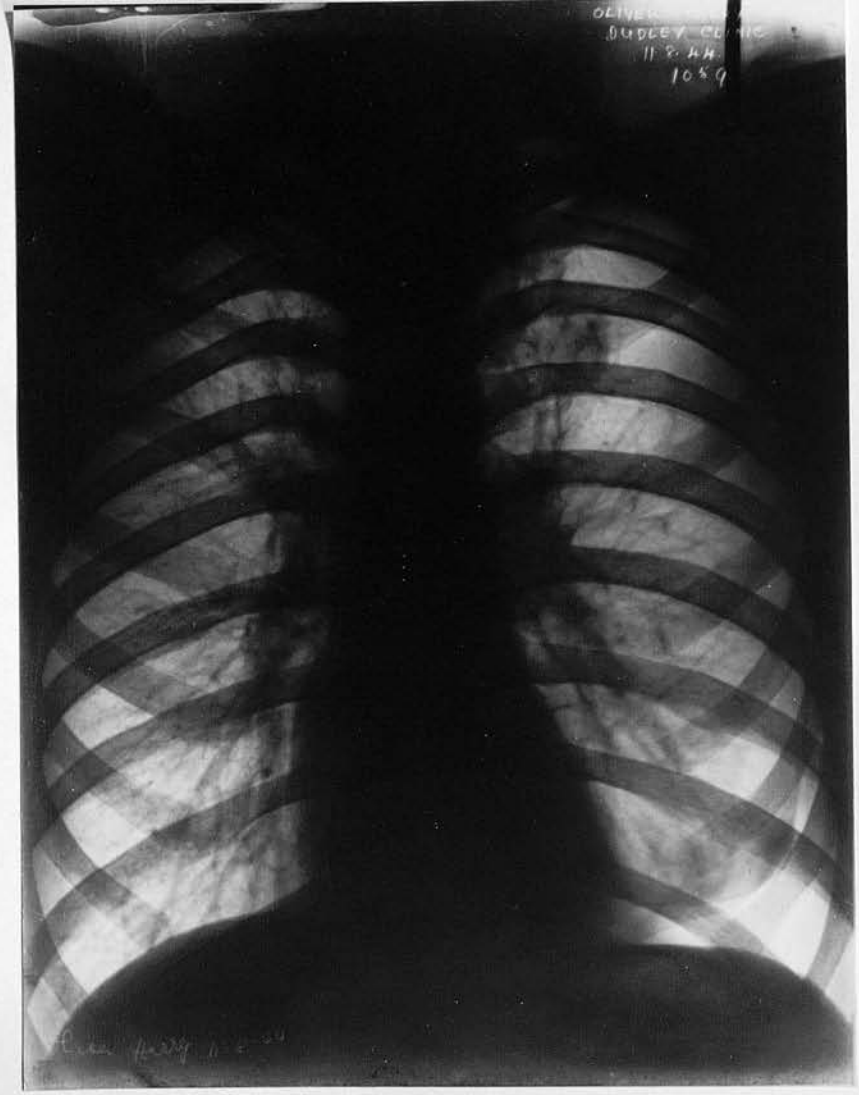


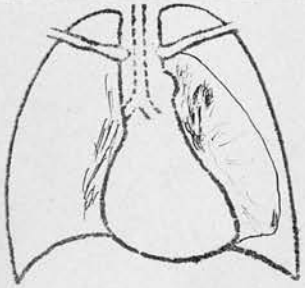


(15·11·43)

PLATE XIX

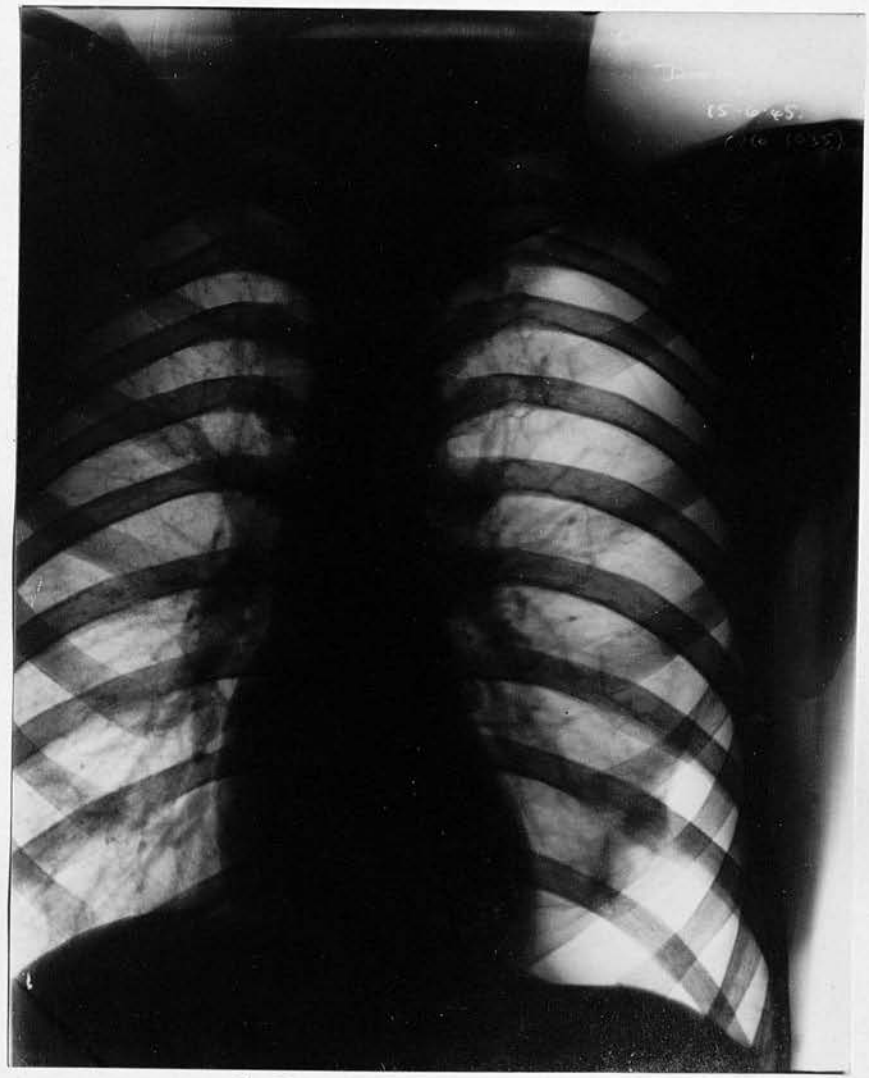
Case IV

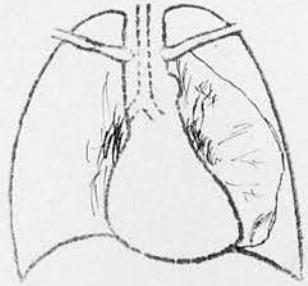




(11 · 8 · 44)

PLATE XX





(15 · 6 · 45)

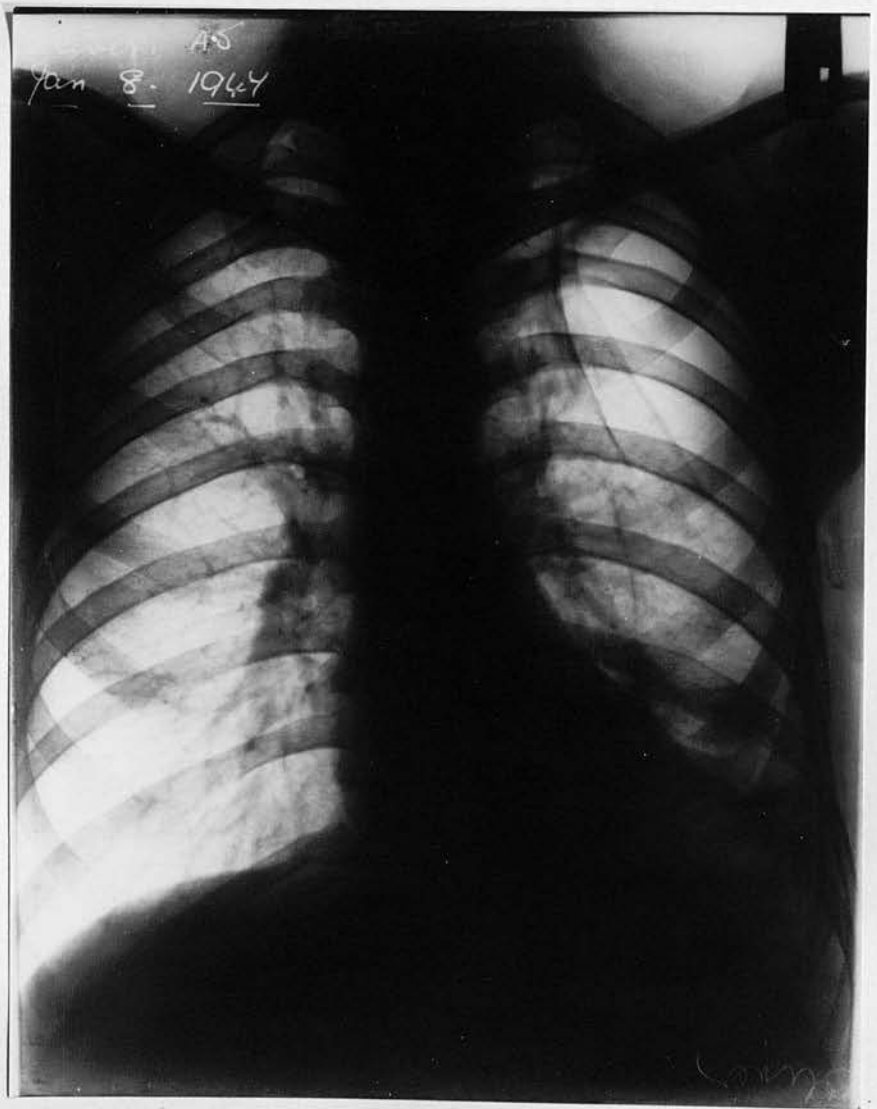
PLATE XXI

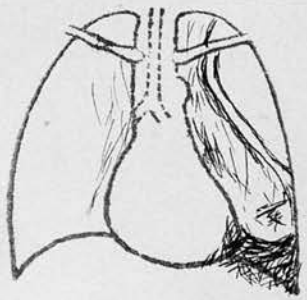
PLATE XIII

(9 m. 2. b)

PLATE XIV

(9 m. m. 01)

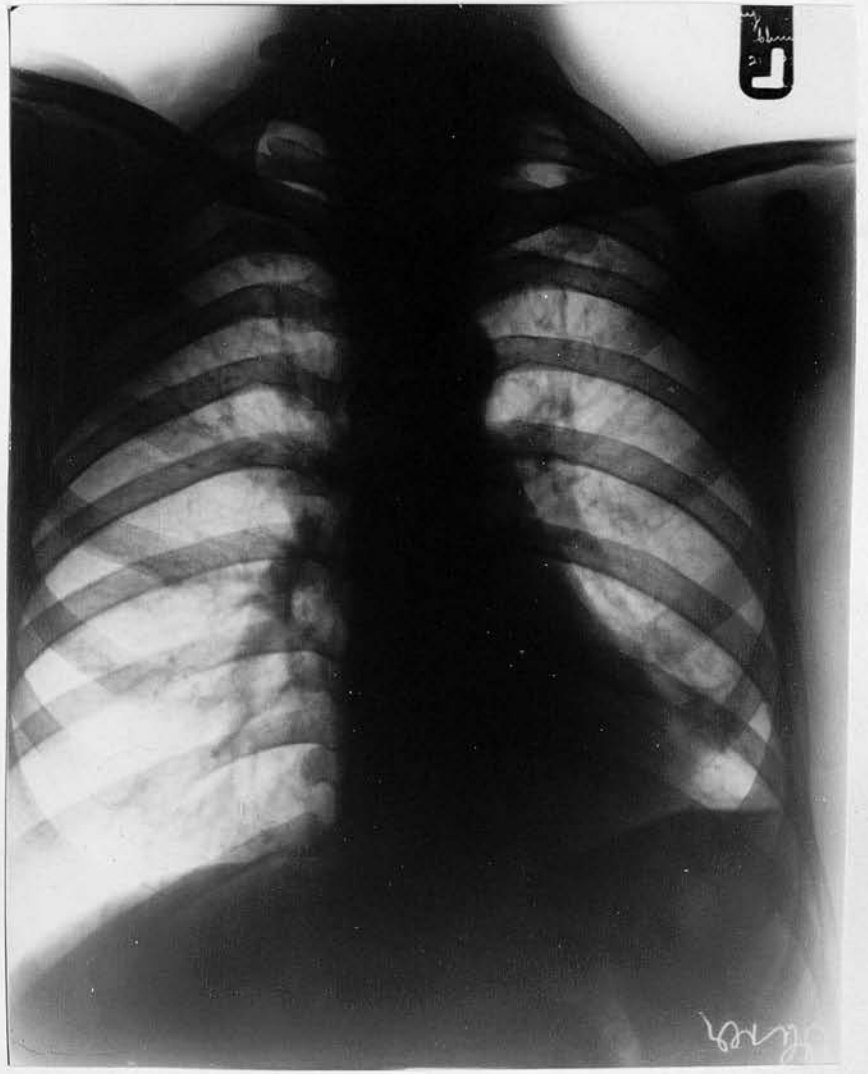


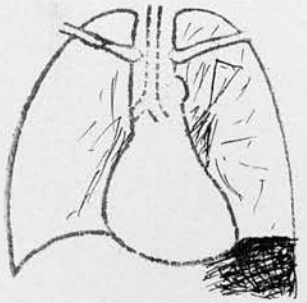


(8.1.47)

Case IV.

PLATE XXIV





(21.10.48)

PLATE XXV

SUMMARY AND CONCLUSIONS.

A cavity is a hole in the lung which is originally the result of necrosis and liquefaction of tissue following invasion by the tubercle bacillus. But a cavity is much more than that: it is the complex product of pathological and mechanical processes which, working in conjunction, are responsible for the behaviour of the cavity once tissue disruption has occurred as a result of necrosis. A cavity cannot be regarded solely in the light of any one of these processes but always as the result of a combination of factors.

A cavity cannot be looked upon as a single unit in itself. Its behaviour is so intimately concerned with the state of the draining bronchus that it cannot be regarded separately from the bronchus. A cavity is really a cavity-bronchus combine. The pathological and mechanical factors mentioned above must be considered from the point of view of their effect upon this combine.

The pathological factors are the results of necrosis, exudation and proliferation. These are the three recognised tissue responses to the tubercle bacillus. Apart from progressive excavation, proliferation plays a leading role in the behaviour of the cavity itself because it results in fibrous tissue which is capable of imparting self-retraction

to the cavity walls, or in non-retracting connective tissue which imparts rigidity. Although the origin and life history of the fibrous tissue is uncertain it appears that some is derived in connection with the epithelioid cells of the tubercle and some is derived as a tissue response to chronic inflammation. With advancing age the fibrous tissue becomes hyalinized and self-retraction of the cavity wall gives place to rigidity. For this reason early treatment of cavities should be sought. These same pathological reactions are to be found in the bronchus when they give rise to mechanical effects within the cavity. These are of the nature of cavity closure or cavity inflation.

The mechanical factors, or forces, are extracavitary and intracavitary. The extracavitary forces are normally due to the negative intrapleural pressure and in consequence are subatmospheric. When in connection with pleural adhesions they may assume formidable and insuperable proportions. The extracavitary forces are conveyed through the lung tissue. The intracavitary forces take the form of intracavitary gas pressures. These pressures might fluctuate with respiratory movements when they are termed dynamic pressures or they may be the mean pressure of the gas contained within the cavity, when they are termed static pressures. The dynamic

intracavitary forces are of relatively little consequence apart from hindering to some extent the healing of the cavity walls. The static intracavitary forces are of paramount importance and generally lead to distension or closure of the cavity. When the bronchus is freely open the static pressure is equal to the pressure of the atmosphere. Pathological changes within the bronchus might result in the lumen becoming completely closed or intermittently closed. Under normal circumstances the lumen is completely open.

If the bronchus is completely open the intracavitary pressure will be atmospheric. Cavity closure will only be secured by removing extracavitary dilating forces, such as pleural adhesions, and permitting the lung to shrink. Even with collapse therapy there is generally a subatmospheric extracavitary force tending to dilate the cavity; thus with an open bronchus complete cavity closure cannot be secured unless by fibrous tissue contraction within the cavity walls. Bronchial occlusion is practically always required in order to secure complete cavity closure.

When the bronchus is occluded the air trapped in the cavity is absorbed into the blood stream. Air absorption is governed by the physical principles of gaseous diffusion. As the

total gaseous pressure of venous blood is 57 m.m. Hg. below the total gaseous pressure of the atmosphere, equilibrium will be reached between the blood circul-:ating in the cavity wall and air trapped within a cavity of which the draining bronchus is blocked, when the intracavitary pressure is - 57 m.m. Hg. This is the lowest intracavitary pressure that can be attained and maintained by natural means. When the bronchus is blocked, the cavity will close by the principle of obstructive atelectasis provided the cavity wall is resilient; otherwise, if the cavity walls are too rigid or are held apart by powerful extracavitary forces as a result of pleural adhesions to the chest wall, the cavity will remain patent with a high negative intracavitary pressure. Absorption of air within a cavity is affected by the thickness and vascularity of the cavity walls and by the amount of caseous deposit and secretions lining them. Air absorption might be very slow in a thick-walled cavity lined by secretions. In a recent cavity with little in the way of an organized wall air absorption might be very rapid, a cavity closing within a matter of hours. When the bronchus is blocked, secretions and caseous matter are also trapped within the cavity. Occasionally these might give rise to troublesome localised and generalised toxaemic symptoms, mainly as a result of secondary

infection. This feature does not however arise sufficiently often to constitute a contraindication to bronchial occlusion being sought as a means of cavity closure.

The bronchial tree acts as an air conduit system to the alveoli. This function is maintained by the physiological movements of the bronchi consisting of inspiratory dilatation with elongation and expiratory contraction with shortening. To these are added the action of the cilia of the mucous membrane. The expiratory contraction of the bronchial walls propels foreign matter towards the upper respiratory tract. Under disease conditions when the lumen of the bronchus is narrowed, the physiological movements of the bronchial walls may result in intermittent occlusion which acts as a check valve mechanism inflating the distal parts of the bronchial tract. This frequently causes inflation of a cavity. The passage of tuberculous infected secretions from a cavity into the bronchial tract causes endobronchial disease by direct contact infection in a large proportion of cases. Evidence suggests that the incidence of bronchial affection in the proximity to the cavity is very high. The broncho-cavitary junction for this reason, and also because of being in different degrees incorporated within the cavity wall itself and therefore subject to the movement and inflammatory reaction of the wall, is particularly liable to act as a check valve.

This fact in conjunction with the extracavitary tension conveyed from the intrapleural space results in the natural tendencies of a tuberculous cavity being towards patency rather than towards closure. A bronchial movement valve of this nature does not create great distension of a cavity owing to the fact that any undue pressure is released with the next inspiratory dilatation of the bronchial lumen. If the mucosal swelling is marked, however, the efficiency of the valve increases until a stage is reached when the valve does not open except with a forced inspiratory movement, when air will be drawn in and trapped, and cannot easily escape. Other types of bronchial and broncho-cavitary valves are described. In those valves where the underlying mechanism is not the movement of the bronchial walls, the effect is due to pressure mechanics on the principle of a ball and socket valve. In these latter the greater the pressure created behind the valve the more efficient that valve becomes. Tension cavities are inflated by small quantities of air at infrequent intervals. The prerequisite to the entry of more air into the cavity is the production of a strong negative intracavitary pressure. This is produced by such conditions as coughing which involve forced inspiratory effort. In view of the physiological movements of the bronchi, the

the prevalence of secretions and foreign matter, and of some degree of endobronchitis in the bronchial tract draining cavities in general, varying degrees of check valve mechanics are extremely common, though sometimes they may only function for a short time. It is not known whether bronchial movements of dilatation and contraction cease at times in the smaller bronchi under conditions of inflammation of their walls. In view of the largely passive nature of their movements it seems unlikely that they often cease altogether under such conditions, especially in the face of forced inspiratory efforts.

The causes for persistence of cavities may be catagorised as extracavitary - generally due to pleural adhesion; intracavitary - usually as a result of a check valve within the bronchus; rigidity of the cavity walls due to chronicity of disease and the deposition of connective tissue. Subpleural cavities form a class by themselves and generally persist because of one or more of the above factors in conjunction with a deficiency of compensatory emplysema. Most cavities persist because bronchial occlusion has not taken place. This may be because of extracavitary factors; on the other hand it may be because of intracavitary factors: it may simply be because tuberculous disease has continued within the bronchus and prevented sealing and healing of the internal bronchial surfaces. If the extracavitary

forces can be removed (adhesion section) or the distension within the cavity be released, the bronchial lumen may be able to occlude. The key site for this to happen is most likely at the broncho-cavitary junction. At this point not only is the maximum bronchial stenosis likely to occur as a result of disease, but further occlusion will take place as the cavity walls shrink together nipping the orifice between them in the process. Frequent involvement at this site helps to explain the presence of a tension cavity without segmental emphysema also being found. It is only when the draining bronchus of a cavity has closed by scar tissue obliteration of its lumen that a cavity can be regarded as closed. Even then reopening might occur as a result of exacerbation of disease within the cavity or its walls, especially when healing has taken place by inspissation of caseous contents, which is the commonest method of cavity healing.

The methods of treating a persistent cavity are very numerous. One of the pitfalls to be avoided is the employment of too many of the available means of treatment. The cause of the persistence of the cavity should be estimated as far as is possible and the line for further treatment decided accordingly. The main feature requiring rectification will in most cases be either adhesions of the visceral pleura to the chest wall or a

bronchial check valve.

Pleural adhesions which cannot be cauterized on thoraeoscopy may necessitate abandoning a pneumothorax and carrying out thoracoplasty. On the other hand extrapleural pneumothorax might be a suit-:able line for further approach. Open pulmolysis as advocated by Hedvall in Sweden (72) gives a very promising new line of approach in cases where the pleural adhesions are too extensive for cauterization. Occasionally the judicious supplementing of a pneumothorax collapse with phrenic paralysis with or without the addition of pneumoperitoneum will provide sufficient relaxation of the lung to effect cavity closure. If this method is to be tried it should not be persisted with too long if it does not show signs of being effective. A matter of a few weeks only should be enough for an indication whether or not the method is going to be effective. The objection to performing phrenic paralysis on such occasions is that if it fails, thoracoplasty may very likely be the next step, and some thoracic surgeons dislike performing this operation in the presence of a paralysed diaphragm on account of lower lobe collapse. Mediastinal adhesions may form an insuperable problem.

Tension cavitation due to a bronchial valve may prove extremely troublesome. In such cases treatment might be directed towards the valve

in an attempt to convert intermittent bronchial occlusion into an open or a completely closed bronchus. Alternatively the valve site may be passed over and cavernostomy or suction drainage be performed, or an attempt might be made to close the bronchus by means of sclerosing or occluding agents.

Occasionally the partial letting up of a pneumothorax collapse or inducing a shallow pneumothorax or supplementing a pneumothorax with phrenic paralysis, with or without pneumoperitoneum, is successful in converting an intermittent bronchial occlusion - or in its stages of advanced stenosis, an occasionally opening bronchus - into an open or closed bronchus. As in the case of phrenic paralysis and pleural adhesions, much time can be wasted by employing these methods. If they are to be tried they should be abandoned at an early date if they show no signs of being really effective. Thoracoplasty for tension cavity is well worth trial. If the cavity is large and surrounded by atelectatic lung, preliminary Monaldi suction drainage is to be recommended. A smaller tension cavity appearing or persisting after thoracoplasty may remain closed following needle aspiration of air, otherwise continuous transthoracic suction might be necessary. Bronchoscopic suction is preferable to transthoracic aspiration as a first method of approach. Thoracoplasty is not suitable in the presence of marked stricture of a large bronchus.

In such cases treatment of the endobronchial disease with streptomycin or else pulmonary resection is indicated. Bronschoscopic suction at repeated intervals has been employed with considerable success in cases of tension cavity under pneumothorax. This method of treatment is based upon sound principles and fuller trial of this means is strongly to be recommended. It may be that a technique using a costo-phrenic bronschoscope and biplane fluoroscopy, as described by Jackson (33) would prove valuable. By this means, visualising the bronchial tree in two planes on the screen, a fine bronchoscope can reach the peripheral bronchi, when suction may be applied. Streptomycin stands high upon the list of means to be employed in the treatment of tension cavities. This antibiotic is as yet on trial but it is to be hoped that it will solve many of the problems which arise in the treatment of persistent cavities in which the underlying cause is tuberculous endobronchial disease. Open cavernostomy as a salvage operation in the hands of some surgeons has proved to be very effective. Attempts to close the bronchus by the use of sclerosing agents have not been successful; the introduction of foreign bodies to act as blocking agents, though rather more successful, have never become popular on account of complications or technical difficulties. Pulmonary resection is becoming increasingly safe as an operation where

other means of treatment are inadvisable or have failed. The underlying cause of tension cavitation is most frequently tuberculous endobronchial disease and treatment will have to be directed towards healing, or removing the effects of, that condition.

For the rigid-walled cavity in chronic fibroid phthisis, thoracoplasty is probably the most satisfactory treatment. Not only is adequate pulmonary relaxation generally afforded but bronchial occlusion as a result of the contraction of scar tissue in and around the bronchus is very likely to be attained.

The cases which have been studied in connection with this thesis have not been demonstrated with a view to indicating the right way of treating persistent cavities. They serve rather as illustrations of what not to do when treating such difficult cases and a large measure of the responsibility for this is accepted by the author. The cases have been studied principally with a view to examining the mechanical and pathological processes underlying such conditions and applying them to the principles established in the main part of the thesis. Criticism in discussing these cases has not been spared. It is only right to point out, however, that satisfactory treatment cannot be given to cases such as these if the means are not available. In the post-war years facilities for thoracic surgery were very deficient in a number of provincial

Sanatoria. For several months during 1946,- the period covered by the four cases in question,- there were no means available for major surgery and for part of that time there were no facilities for minor procedures such as phrenic paralysis and thoracoscopy. A good deal of time was lost in consequence. The correct and expeditious treatment of cavities in pulmonary tuberculosis can only be carried out when efficient, reliable and up-to-date means of full investigation are available and when the necessary surgical treatment is at hand. These however do not effect the exercise of right judgment in dealing with the cases, which may only be acquired through experience.

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